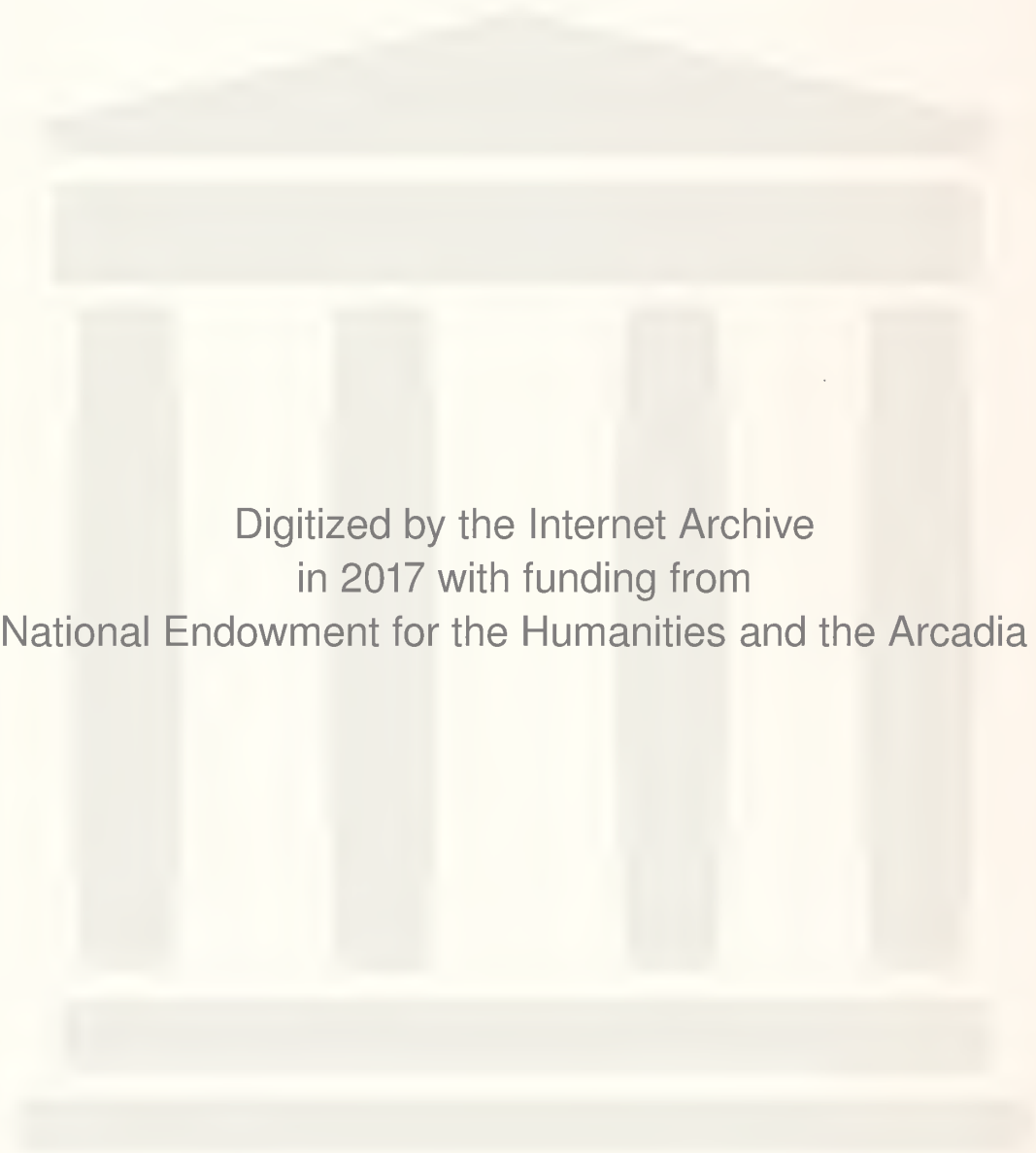

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Rocky Mountain Medical Journal

January - February 1977
Volume 74, No. 1

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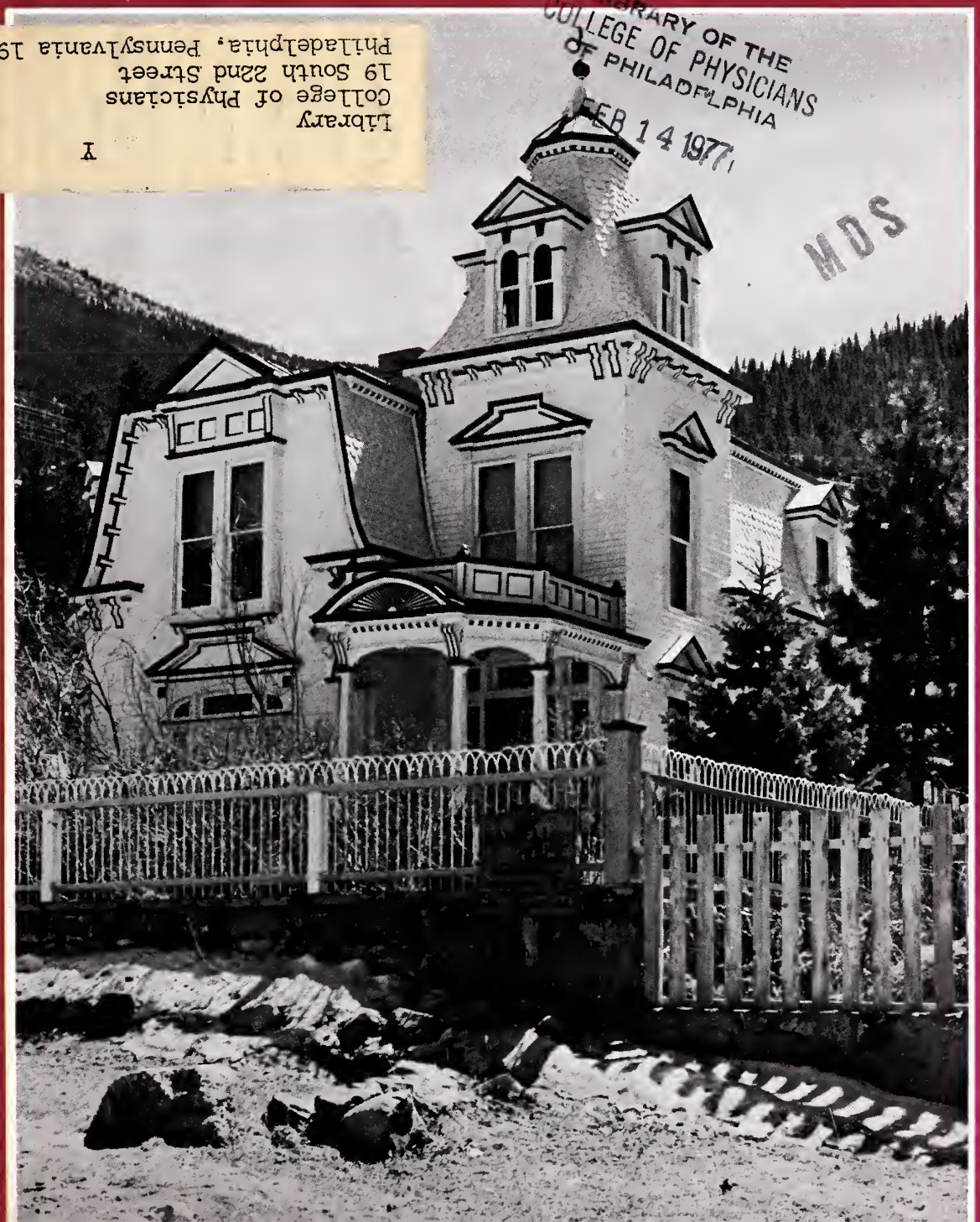
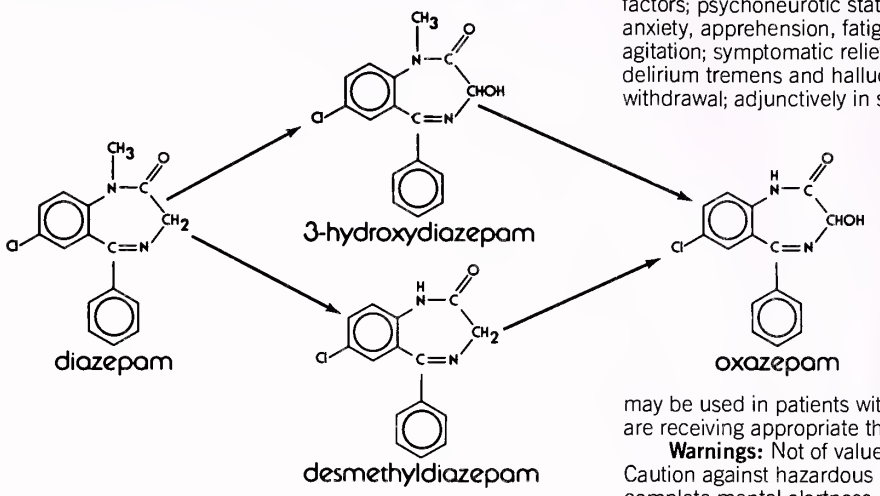


A pharmacokinetic character all its own



Valium (diazepam) is a benzodiazepine with a distinctive pharmacokinetic profile

The pharmacokinetic profile of Valium is one of the characteristics that sets it apart from other benzodiazepines. Consider, in particular, the metabolic pathway of Valium. The three major metabolites of Valium exhibit significant pharmacologic activity—and so, of course, does the parent substance—diazepam itself. All combine to produce the characteristic clinical response seen with Valium. The response you have come to know, to want and to trust.

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2-mg, 5-mg, 10-mg scored tablets

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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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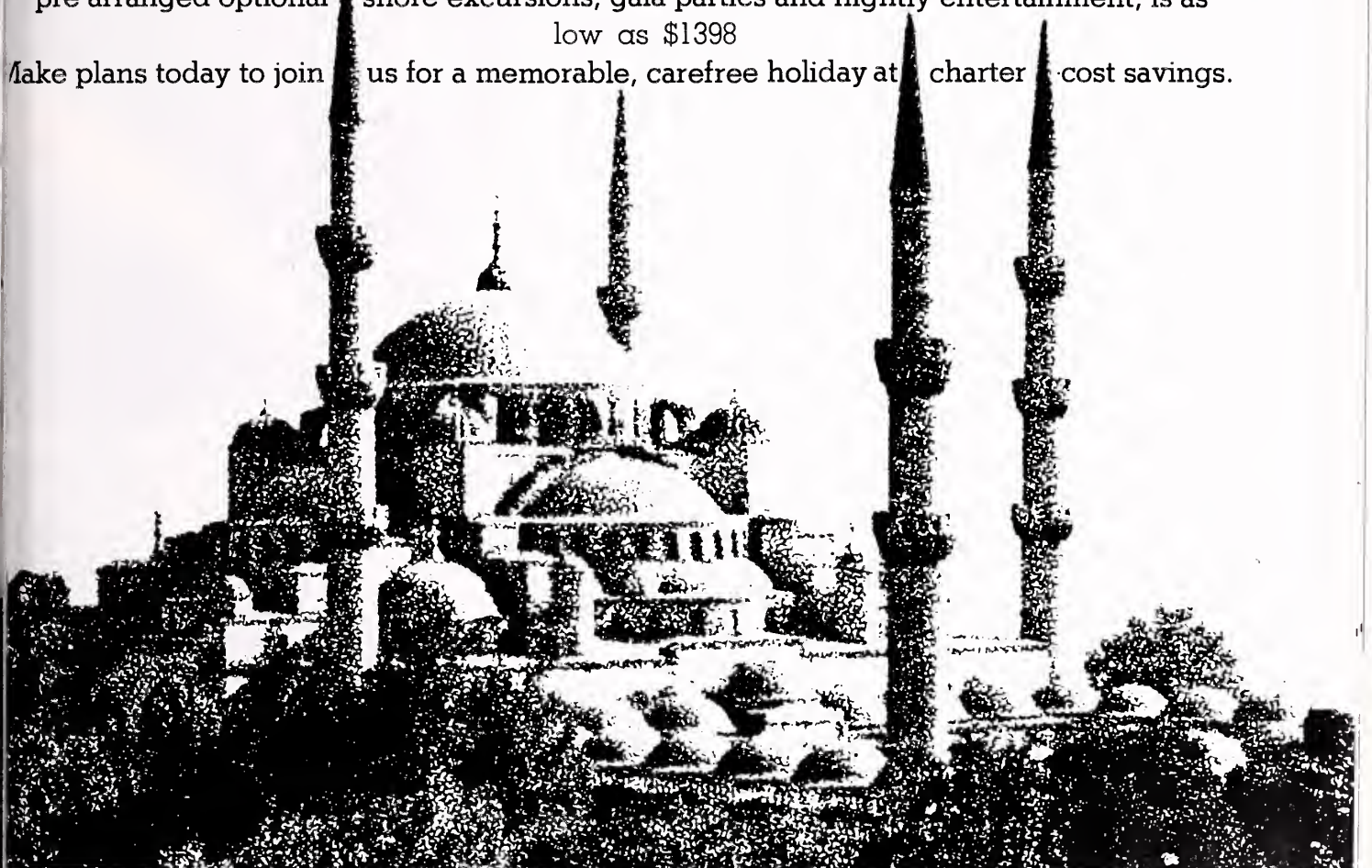

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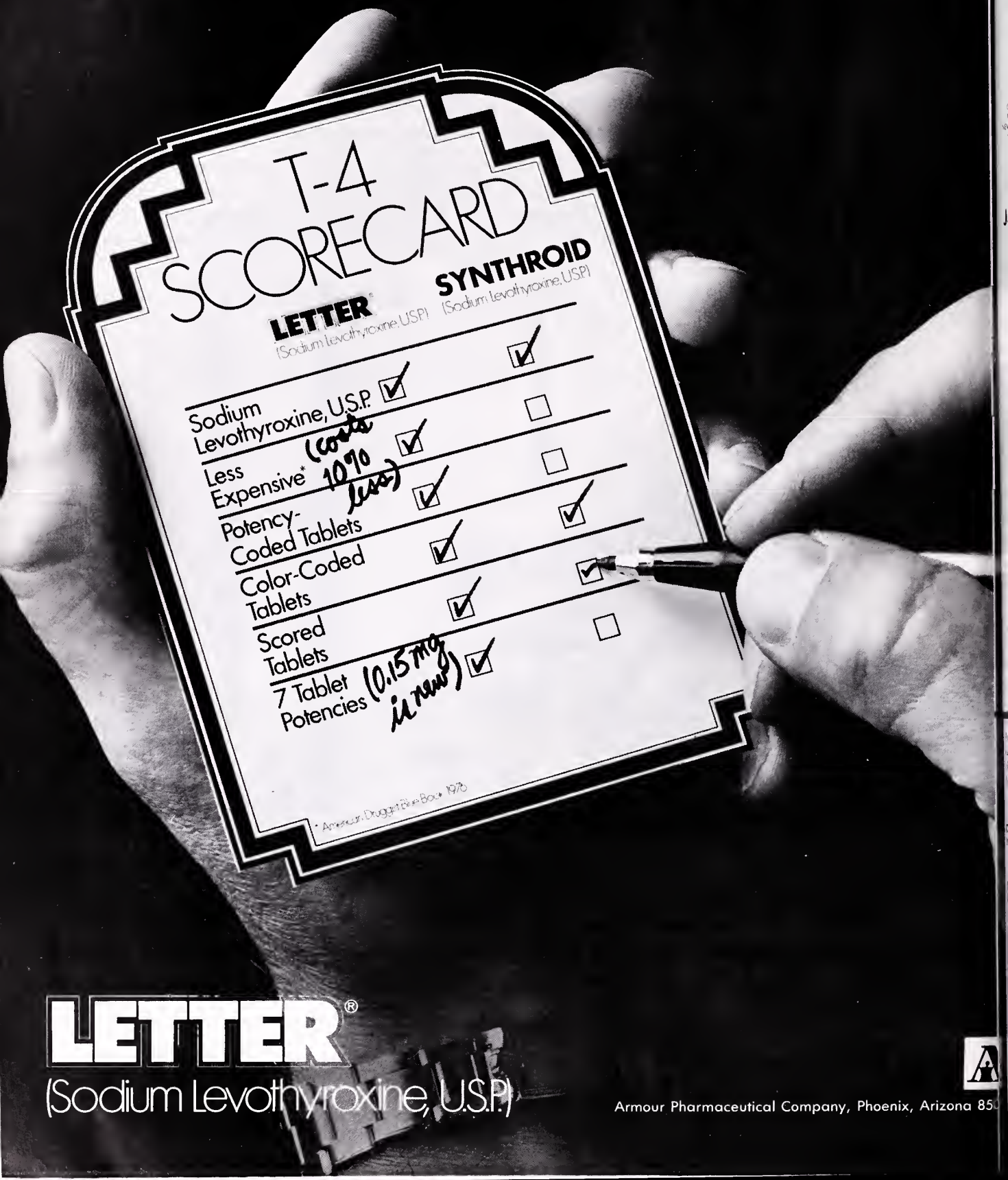

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Each tablet contains 130 mg theophylline, 24 mg ephedrine hydrochloride, and 8 mg phenobarbital.

Each tablet contains 180 mg anhydrous theophylline (90 mg in the immediate release layer and 90 mg in the sustained release layer); 48 mg ephedrine hydrochloride (16 mg in the immediate release layer and 32 mg in the sustained release layer); and 25 mg phenobarbital in the immediate release layer.

SUSTAINED ACTION

Each 5 ml teaspoonful contains 32.5 mg theophylline, 6 mg ephedrine HCl, and 2 mg phenobarbital; the alcohol content is 15%.

See next page for brief summary.

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TEDRAL® Elixir

CAUTION: Federal law prohibits dispensing Tedral SA without prescription.

Description. Tedral: each tablet contains 130 mg theophylline, 24 mg ephedrine hydrochloride, and 8 mg phenobarbital.

Tedral SA: each tablet contains 180 mg anhydrous theophylline (90 mg in the immediate release layer and 90 mg in the sustained release layer); 48 mg ephedrine hydrochloride (16 mg in the immediate release layer and 32 mg in the sustained release layer); 25 mg phenobarbital in the immediate release layer.

Tedral Elixir: each 5 ml teaspoonful contains 32.5 mg theophylline, 6 mg ephedrine hydrochloride, and 2 mg phenobarbital; the alcohol content is 15%.

Indications. Tedral, Tedral SA, and Tedral Elixir are indicated for the symptomatic relief of bronchial asthma, asthmatic bronchitis, and other bronchospastic disorders. They may also be used prophylactically to abort or minimize asthmatic attacks and are of value in managing occasional, seasonal or perennial asthma.

Tedral SA (Sustained Action) offers the convenience of b.i.d. dosage.

Tedral Elixir is convenient for persons who may have difficulty in swallowing tablets.

These Tedral formulations are adjuncts in the total management of the asthmatic patient. Acute or severe asthmatic attacks may necessitate supplemental therapy with other drugs by inhalation or other parenteral routes.

Contraindications. Sensitivity to any of the ingredients; porphyria.

Warnings. Drowsiness may occur. PHENOBARBITAL MAY BE HABIT-FORMING.

Precautions. Use with caution in the presence of cardiovascular disease, severe hypertension, hyperthyroidism, prostatic hypertrophy, or glaucoma.

Adverse Reactions. Mild epigastric distress, palpitation, tremulousness, insomnia, difficulty of micturition, and CNS stimulation have been reported.

Average Dosage. *Prophylactic or Therapeutic.*

Tedral: *Adults*—One or two tablets every 4 hours. *Children*—(Over 60 lb) one-half the adult dose.

Tedral SA: *Adults*—One tablet on arising and one tablet 12 hours later. Tablets should not be chewed. *Children*—Not established for children under 12.

Tedral Elixir: Note: One teaspoonful is equivalent to *one-quarter* Tedral tablet. *Children*—One teaspoonful per 30 lb body weight, every 4-6 hours, unless prescribed otherwise by physician. Should be given to children under 2 years of age only with extreme caution. *Adults*—One to two tablespoonfuls every four hours.

Supplied. Tedral: White, uncoated scored tablets in bottles of 24 (N 0047-0230-24), 100 (N 0047-0230-51) and 1000 (N 0047-0230-60). Also in Unit Dose—package of 10 x 10 strips (N 0047-0230-11).

Tedral SA: Double-layered, uncoated, coral/mottled white tablets in bottles of 100 (N 0047-0231-51) and 1000 (N 0047-0231-60). Also in Unit Dose—package of 10 x 10 strips (N 0047-0231-11).

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Full information is available on request.

in Brief

A summary of AMA, medical & health news

American Medical Association / 535 North Dearborn Street
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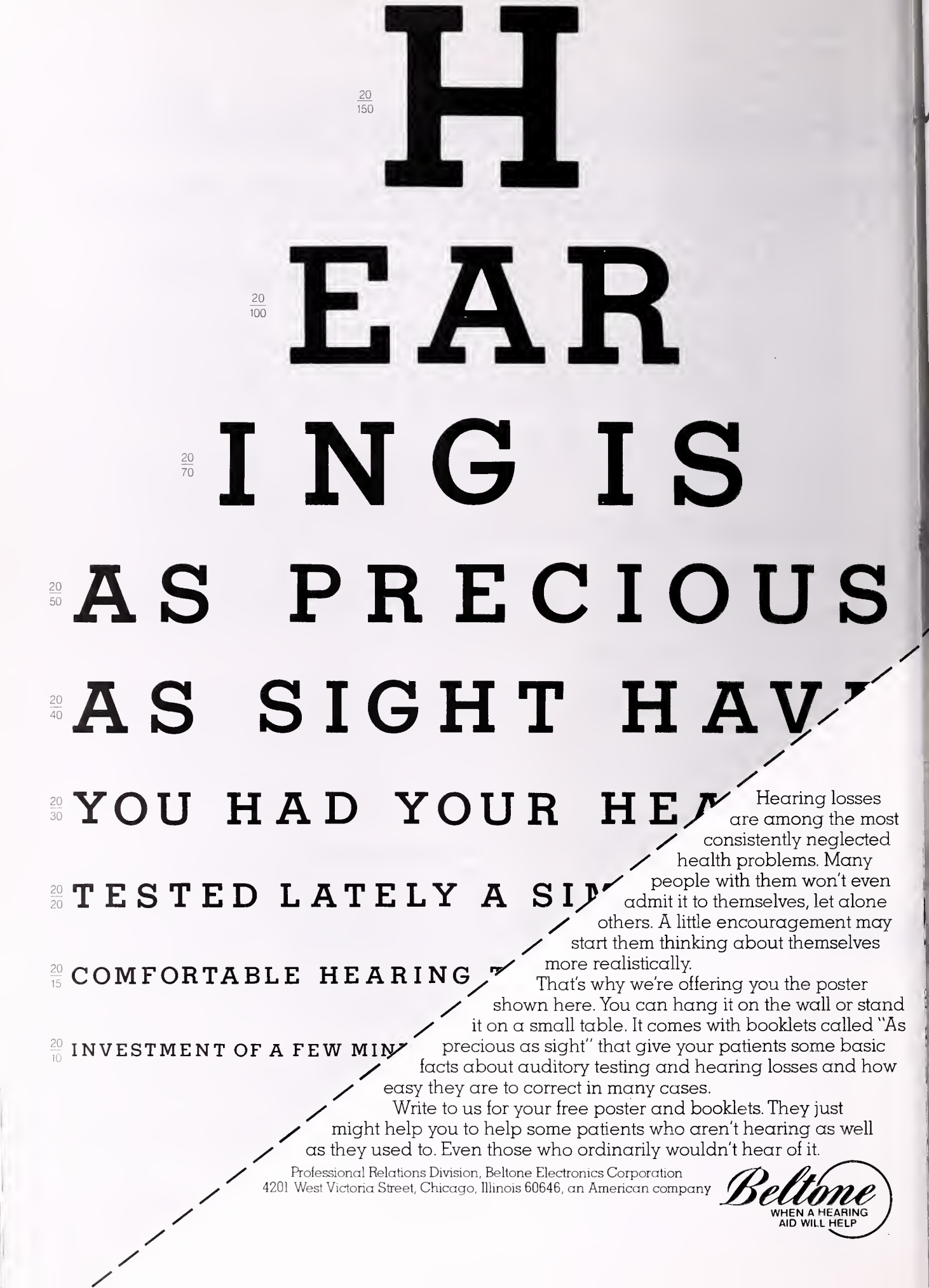
HEW's Medical Services Administration, in a memo to the states, encouraged adoption of the AMA uniform health insurance claim form by all state Medicaid agencies. The agency said the AMA form would meet most Medicaid requirements. The form was developed by the Council on Medical Service in response to a House of Delegates action calling for a means of reducing paperwork and simplifying billing.

The AMA offered assistance and cooperation to the Medicaid Task Force of the National Governors Conference in its efforts to develop proposals to improve the program. The task force, headed by Gov. George Busbee of Georgia, included representatives from 11 other states. The group will present its recommendations to the National Governors Conference in February. Federal legislation aimed at abuses of the Medicaid program is expected to be introduced early in the 95th Congress.

"A Critical Difference," a 60-minute documentary film produced by the AMA for the general TV viewing audience, has been shown in eight cities on network-affiliated stations to approximately 400,000 viewers. The film explores various aspects of health care in America and contrasts federally run health care programs with physician control of patient care.

The AMA announced a grant of $25,000 to the National Citizens Committee for Broadcasting, a media reform group based in Washington, D.C., concerned with documenting the amount of television violence portrayed in prime time network television. AMA EVP James H. Sammons, MD, said the grant "represents a strong commitment by the AMA to endorse and finance activities that will encourage the industry to reduce the amount of violence in TV programming."

The AMA's Reinsurance Company--American Medical Assurance Co.--officially went into business with the signing of a service agreement with the Kemper Reinsurance Co. Kemper will handle AMACO's accounting, taxes and insurance commission statements, provide underwriting consultation, and manage investments. AMACO will provide reinsurance for qualifying physician-owned medical liability companies.



Hearing losses are among the most consistently neglected health problems. Many people with them won't even admit it to themselves, let alone others. A little encouragement may start them thinking about themselves more realistically.

That's why we're offering you the poster shown here. You can hang it on the wall or stand it on a small table. It comes with booklets called "As precious as sight" that give your patients some basic facts about auditory testing and hearing losses and how easy they are to correct in many cases.

Write to us for your free poster and booklets. They just might help you to help some patients who aren't hearing as well as they used to. Even those who ordinarily wouldn't hear of it.

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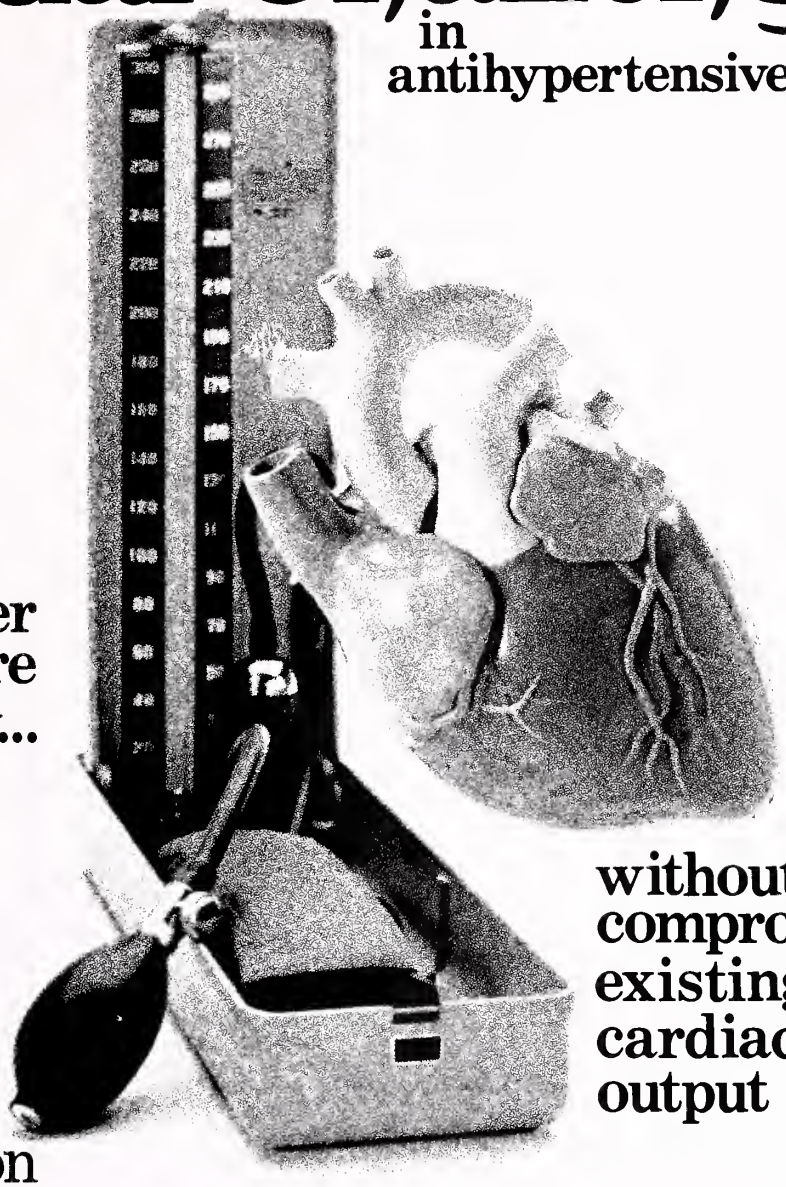

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blood pressure
effectively...

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existing
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DOMET is contraindicated in active hepatic disease, hypersensitivity to the drug, and if evious methyldopa therapy has been associated with liver disorders.
s important to recognize that a positive Coombs test, hemolytic anemia, and liver disorders ay occur with methyldopa therapy. The rare occurrences of hemolytic anemia or liver disorders uld lead to potentially fatal complications unless properly recognized and managed. For more tails see the brief summary of prescribing information.

r a brief summary of prescribing information, please see following page.

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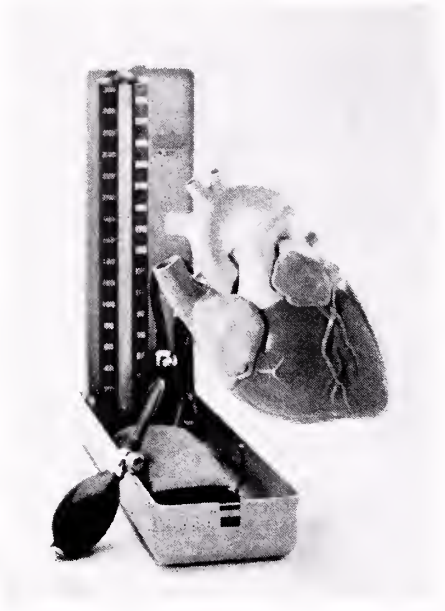

in hypertension

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(METHYLDOPA|MSD)

helps lower blood pressure effectively... usually with no direct effect on cardiac function– cardiac output is usually maintained

Contraindications: Active hepatic disease, such as acute hepatitis and active cirrhosis; if previous methyldopa therapy has been associated with liver disorders (see Warnings); hypersensitivity.

Warnings: It is important to recognize that a positive Coombs test, hemolytic anemia, and liver disorders may occur with methyldopa therapy. The rare occurrences of hemolytic anemia or liver disorders could lead to potentially fatal complications unless properly recognized and managed. Read this section carefully to understand these reactions.

With prolonged methyldopa therapy, 10% to 20% of patients develop a positive direct Coombs test, usually between 6 and 12 months of therapy. Lowest incidence is at daily dosage of 1 g or less. This on rare occasions may be associated with hemolytic anemia, which could lead to potentially fatal complications. One cannot predict which patients with a positive direct Coombs test may develop hemolytic anemia. Prior existence or development of a positive direct Coombs test is not in itself a contraindication to use of methyldopa. If a positive Coombs test develops during methyldopa therapy, determine whether hemolytic anemia exists and whether the positive Coombs test may be a problem. For example, in addition to a positive direct Coombs test there is less often a positive indirect Coombs test which may interfere with cross matching of blood.

At the start of methyldopa therapy, it is desirable to do a blood count (hematocrit, hemoglobin, or red cell count) for a baseline or to establish whether there is anemia. Periodic blood counts should be done during therapy to detect hemolytic anemia. It may be useful to do a direct Coombs test before therapy and at 6 and 12 months after the start of therapy. If Coombs-positive hemolytic anemia occurs, the cause may be methyldopa and the drug should be discontinued. Usually the anemia remits promptly. If not, corticosteroids may be given and other causes of anemia should be considered. If the hemolytic anemia is related to methyldopa, the drug should not be reinstituted. When methyldopa causes Coombs positivity alone or with hemolytic anemia, the red cell is usually coated with gamma globulin of the IgG (gamma G) class only. The positive Coombs test may not revert to normal until weeks to months after methyldopa is stopped.

Should the need for transfusion arise in a patient receiving methyldopa, both a direct and an indirect Coombs test should be performed on his blood. In the absence of hemolytic anemia, usually only the direct Coombs test will be positive. A positive direct Coombs test alone will not interfere with typing or cross matching. If the indirect Coombs test is also positive, problems may arise in the major cross match and the assistance of a hematologist or transfusion expert will be needed.

Fever has occurred within first 3 weeks of therapy, sometimes with eosinophilia or abnormalities in liver function tests, such as serum alkaline phosphatase, serum transaminases (SGOT, SGPT), bilirubin, cephalin cholesterol flocculation, prothrombin time, and bromsulphalein retention. Jaundice, with or without fever, may occur, with onset usually in the first 2 to 3 months of therapy. In some patients the findings are consistent with those of cholestasis. Rarely fatal hepatic necrosis has been reported. These hepatic changes may represent hypersensitivity reactions; periodic determination of hepatic function should be done particularly during the first 6 to 12 weeks of therapy or whenever an unexplained fever occurs. If fever and abnormalities in liver function tests or jaundice appear, stop therapy with methyldopa. If caused by methyldopa, the temperature and abnormalities in liver function characteristically have reverted to normal when the drug was discontinued. Methyldopa should not be reinstituted in such patients.

Rarely, a reversible reduction of the white blood cell count with primary effect on granulocytes has been seen. Reversible thrombocytopenia has occurred rarely. When used with other antihypertensive drugs, potentiation of antihypertensive effect may occur. Patients should be followed carefully to detect side reactions or unusual manifestations of drug idiosyncrasy.

Use in Pregnancy: Use of any drug in women who are or may become pregnant requires that anticipated benefits be weighed against possible risks; possibility of fetal injury can not be excluded.

Precautions: Should be used with caution in patients with history of previous liver disease or dysfunction (see Warnings). May interfere with measurement of: uric acid by the phosphotungstate method, creatinine by the alkaline picrate method, and SGOT by colorimetric methods. Since methyldopa causes fluorescence in urine samples at the same wavelengths as catecholamines, falsely high levels of urinary catecholamines may be reported. This will interfere with the diagnosis of pheochromocytoma. It is important to recognize this phenomenon before a patient with a possible pheochromocytoma is subjected to surgery. Methyldopa is not recommended for patients with pheochromocytoma. Urine exposed to air after voiding may darken because of breakdown of methyldopa or its metabolites.

Stop drug if involuntary choreoathetotic movemen occur in patients with severe bilateral cerebrovasc lar disease. Patients may require reduced doses anesthetics; hypotension occurring during ane thesia usually can be controlled with vasopressor Hypertension has recurred after dialysis in patien on methyldopa because the drug is removed by th procedure.

Adverse Reactions: *Central nervous syste* Sedation, headache, asthenia or weakness, usual early and transient; dizziness, lightheadednes symptoms of cerebrovascular insufficienc paresthesias, parkinsonism, Bell's palsy, decrease mental acuity, involuntary choreoathetotic mov ments; psychic disturbances, including nightmare and reversible mild psychoses or depression.

Cardiovascular: Bradycardia, aggravation of angin pectoris. Orthostatic hypotension (decrease dai dosage). Edema (and weight gain) usually relieve by use of a diuretic. (Discontinue methyldopa edema progresses or signs of heart failure appear

Gastrointestinal: Nausea, vomiting, distention, co stipation, flatus, diarrhea, mild dryness of mouth, so or "black" tongue, pancreatitis, sialadenitis.

Hepatic: Abnormal liver function tests, jaundic liver disorders.

Hematologic: Positive Coombs test, hemolyt anemia. Leukopenia, granulocytopenia, throm bocytopenia.

Allergic: Drug-related fever, myocarditis.

Other: Nasal stuffiness, rise in BUN, breast enlarg ment, gynecomastia, lactation, impotence, decrease libido, dermatologic reactions including eczema a lichenoid eruptions, mild arthralgia, myalgia.

Note: Initial adult dosage should be limited t 500 mg daily when given with antihypertensive other than thiazides. Tolerance may occur, usual between second and third month of therapy; i creased dosage or adding a thiazide frequent restores effective control. Patients with impaire renal function may respond to smaller doses. Sy cope in older patients may be related to increase sensitivity and advanced arteriosclerotic vascula disease; this may be avoided by lower doses.

How Supplied: Tablets, containing 125 m methyldopa each, in bottles of 100; Tablets, contain ing 250 mg methyldopa each, in single-uni packages of 100 and bottles of 100 and 1000 Tablets, containing 500 mg methyldopa each, i single-unit packages of 100 and bottles of 100.

For more detailed information, consult your MS representative or see full prescribing informa tion. Merck Sharp & Dohme, Division of Merck Co., Inc., West Point, Pa. 19486 J6AM07 (707

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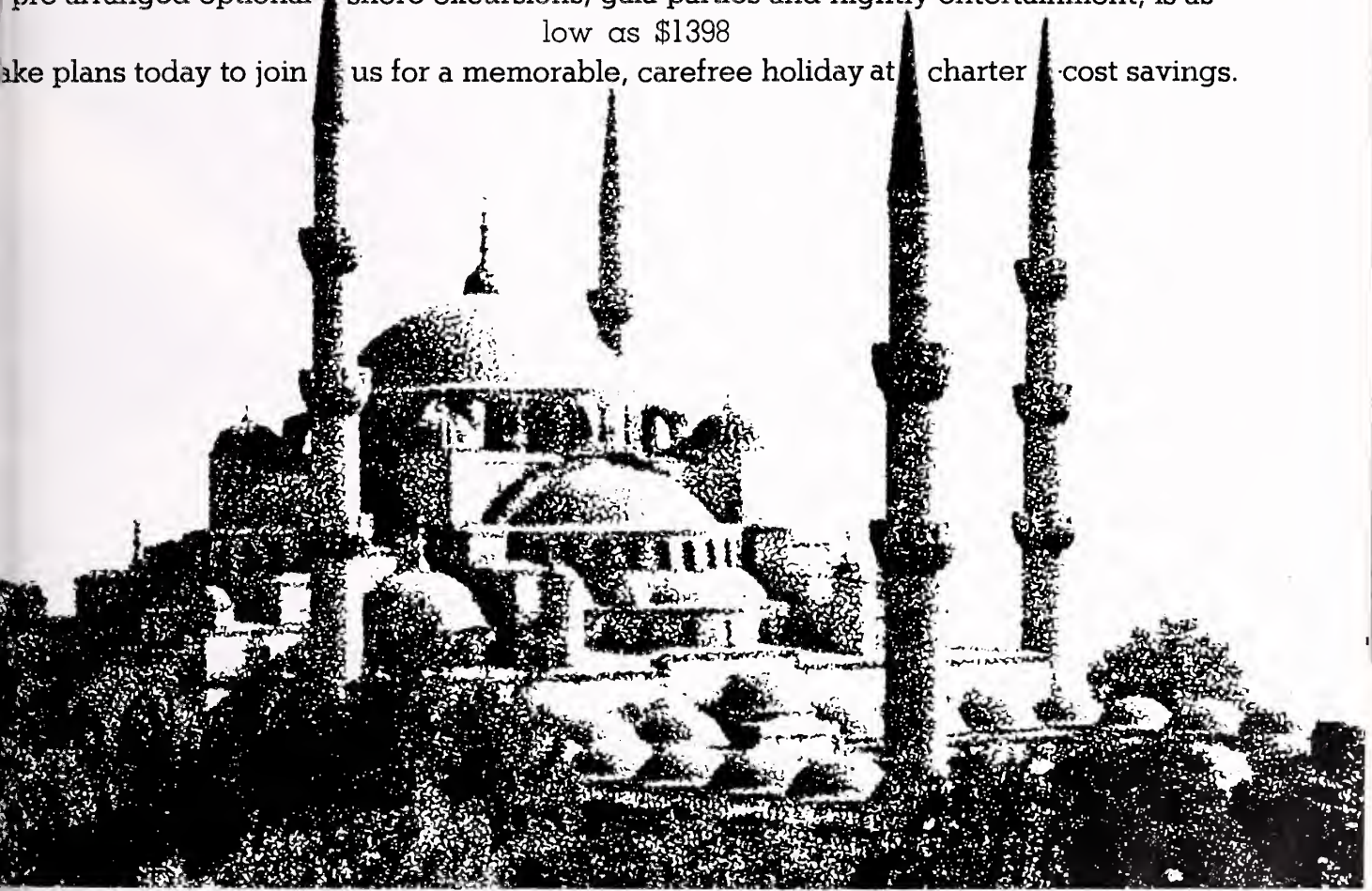

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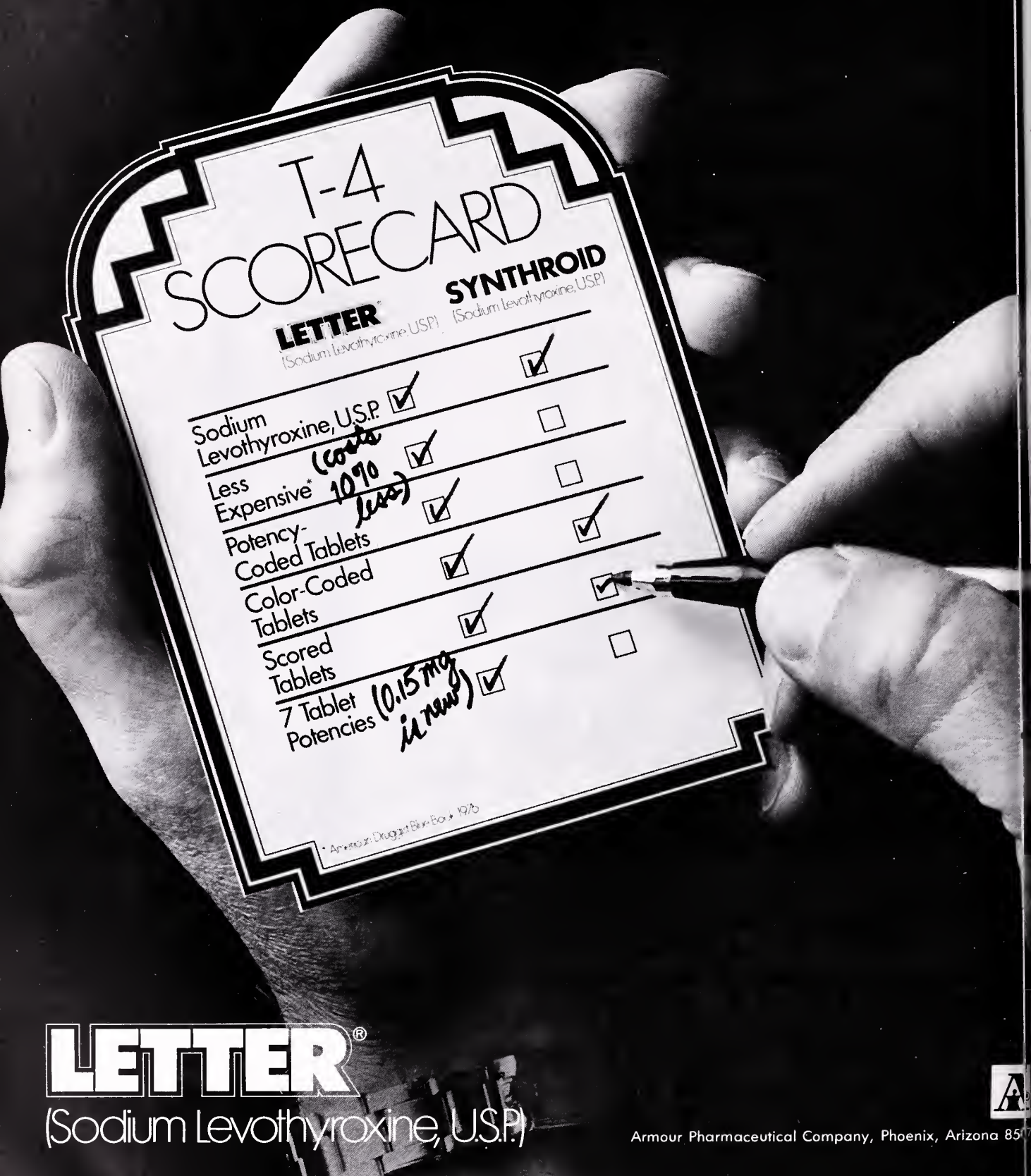

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Rocky Mountain Medical Journal
COLORADO MONTANA NEW MEXICO WYOMING

January - February 1977
VOLUME 74 - NUMBER 1

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Each 5 ml teaspoonful contains 32.5 mg theophylline, 6 mg ephedrine HCl, and 2 mg phenobarbital; the alcohol content is 15%.

See next page for brief summary.

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Warnings. Drowsiness may occur. PHENOBARBITAL MAY BE HABIT-FORMING.

Precautions. Use with caution in the presence of cardiovascular disease, severe hypertension, hyperthyroidism, prostatic hypertrophy, or glaucoma.

Adverse Reactions. Mild epigastric distress, palpitation, tremulousness, insomnia, difficulty of micturition, and CNS stimulation have been reported.

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Tedral: *Adults*—One or two tablets every 4 hours. *Children*—(Over 60 lb) one-half the adult dose.

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in Brief

A summary of AMA, medical & health news

American Medical Association / 535 North Dearborn Street
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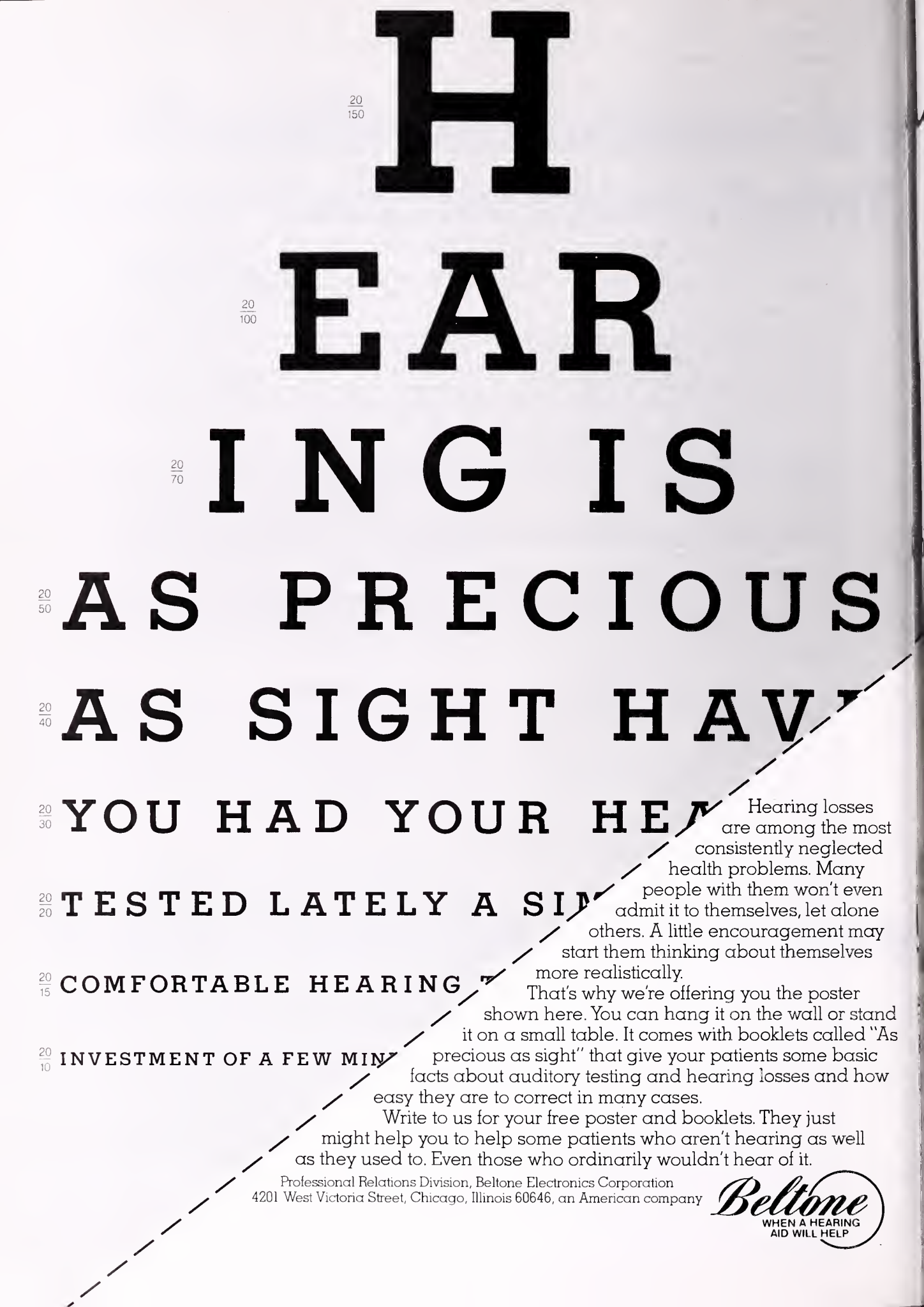
HEW's Medical Services Administration, in a memo to the states, encouraged adoption of the AMA uniform health insurance claim form by all state Medicaid agencies. The agency said the AMA form would meet most Medicaid requirements. The form was developed by the Council on Medical Service in response to a House of Delegates action calling for a means of reducing paperwork and simplifying billing.

The AMA offered assistance and cooperation to the Medicaid Task Force of the National Governors Conference in its efforts to develop proposals to improve the program. The task force, headed by Gov. George Busbee of Georgia, included representatives from 11 other states. The group will present its recommendations to the National Governors Conference in February. Federal legislation aimed at abuses of the Medicaid program is expected to be introduced early in the 95th Congress.

"A Critical Difference," a 60-minute documentary film produced by the AMA for the general TV viewing audience, has been shown in eight cities on network-affiliated stations to approximately 400,000 viewers. The film explores various aspects of health care in America and contrasts federally run health care programs with physician control of patient care.

The AMA announced a grant of $25,000 to the National Citizens Committee for Broadcasting, a media reform group based in Washington, D.C., concerned with documenting the amount of television violence portrayed in prime time network television. AMA EVP James H. Sammons, MD, said the grant "represents a strong commitment by the AMA to endorse and finance activities that will encourage the industry to reduce the amount of violence in TV programming."

The AMA's Reinsurance Company--American Medical Assurance Co.--officially went into business with the signing of a service agreement with the Kemper Reinsurance Co. Kemper will handle AMACO's accounting, taxes and insurance commission statements, provide underwriting consultation, and manage investments. AMACO will provide reinsurance for qualifying physician-owned medical liability companies.



Hearing losses are among the most consistently neglected health problems. Many people with them won't even admit it to themselves, let alone others. A little encouragement may start them thinking about themselves more realistically.

That's why we're offering you the poster shown here. You can hang it on the wall or stand it on a small table. It comes with booklets called "As precious as sight" that give your patients some basic facts about auditory testing and hearing losses and how easy they are to correct in many cases.

Write to us for your free poster and booklets. They just might help you to help some patients who aren't hearing as well as they used to. Even those who ordinarily wouldn't hear of it.

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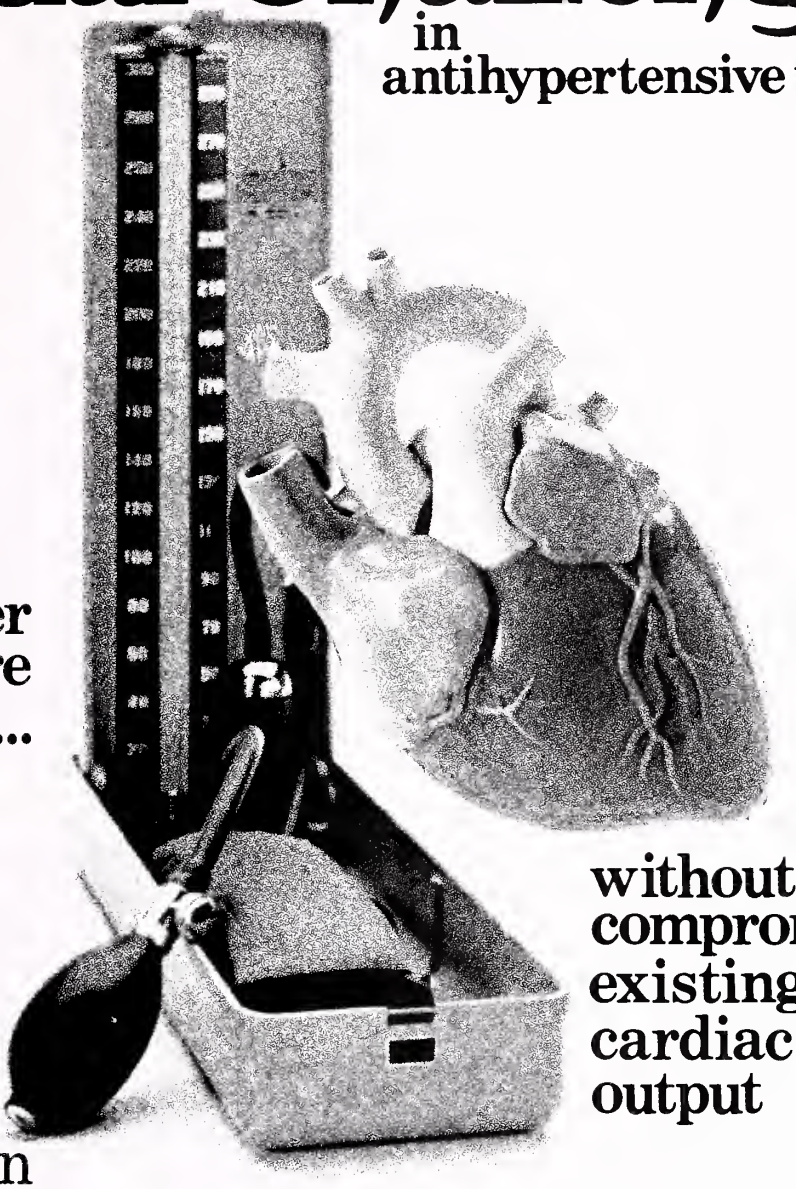
WHEN A HEARING AID WILL HELP

A Dual Challenge

in antihypertensive therapy



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without compromising existing cardiac output

n hypertension

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usually maintained

OMET is contraindicated in active hepatic disease, hypersensitivity to the drug, and if vious methyldopa therapy has been associated with liver disorders.
important to recognize that a positive Coombs test, hemolytic anemia, and liver disorders occur with methyldopa therapy. The rare occurrences of hemolytic anemia or liver disorders ld lead to potentially fatal complications unless properly recognized and managed. For more ails see the brief summary of prescribing information.

a brief summary of prescribing information, please see following page.

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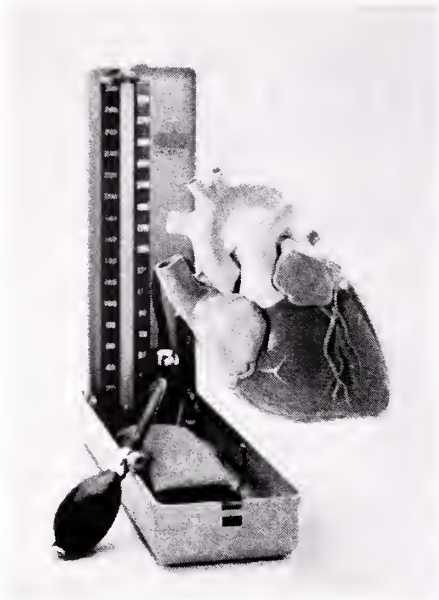

in hypertension

ALDOMET®

(METHYLDOPA|MSD)

helps lower blood pressure effectively... usually with no direct effect on cardiac function– cardiac output is usually maintained

Contraindications: Active hepatic disease, such as acute hepatitis and active cirrhosis; if previous methyldopa therapy has been associated with liver disorders (see Warnings); hypersensitivity.

Warnings: It is important to recognize that a positive Coombs test, hemolytic anemia, and liver disorders may occur with methyldopa therapy. The rare occurrences of hemolytic anemia or liver disorders could lead to potentially fatal complications unless properly recognized and managed. Read this section carefully to understand these reactions.

With prolonged methyldopa therapy, 10% to 20% of patients develop a positive direct Coombs test, usually between 6 and 12 months of therapy. Lowest incidence is at daily dosage of 1 g or less. This on rare occasions may be associated with hemolytic anemia, which could lead to potentially fatal complications. One cannot predict which patients with a positive direct Coombs test may develop hemolytic anemia. Prior existence or development of a positive direct Coombs test is not in itself a contraindication to use of methyldopa. If a positive Coombs test develops during methyldopa therapy, determine whether hemolytic anemia exists and whether the positive Coombs test may be a problem. For example, in addition to a positive direct Coombs test there is less often a positive indirect Coombs test which may interfere with cross matching of blood.

At the start of methyldopa therapy, it is desirable to do a blood count (hematocrit, hemoglobin, or red cell count) for a baseline or to establish whether there is anemia. Periodic blood counts should be done during therapy to detect hemolytic anemia. It may be useful to do a direct Coombs test before therapy and at 6 and 12 months after the start of therapy. If Coombs-positive hemolytic anemia occurs, the cause may be methyldopa and the drug should be discontinued. Usually the anemia remits promptly. If not, corticosteroids may be given and other causes of anemia should be considered. If the hemolytic anemia is related to methyldopa, the drug should not be reinstituted. When methyldopa causes Coombs positivity alone or with hemolytic anemia, the red cell is usually coated with gamma globulin of the IgG (gamma G) class only. The positive Coombs test may not revert to normal until weeks to months after methyldopa is stopped.

Should the need for transfusion arise in a patient receiving methyldopa, both a direct and an indirect Coombs test should be performed on his blood. In the absence of hemolytic anemia, usually only the direct Coombs test will be positive. A positive direct Coombs test alone will not interfere with typing or cross matching. If the indirect Coombs test is also positive, problems may arise in the major cross match and the assistance of a hematologist or transfusion expert will be needed.

Fever has occurred within first 3 weeks of therapy, sometimes with eosinophilia or abnormalities in liver function tests, such as serum alkaline phosphatase, serum transaminases (SGOT, SGPT), bilirubin, cephalin cholesterol flocculation, prothrombin time, and bromsulphalein retention. Jaundice, with or without fever, may occur, with onset usually in the first 2 to 3 months of therapy. In some patients the findings are consistent with those of cholestasis. Rarely fatal hepatic necrosis has been reported. These hepatic changes may represent hypersensitivity reactions; periodic determination of hepatic function should be done particularly during the first 6 to 12 weeks of therapy or whenever an unexplained fever occurs. If fever and abnormalities in liver function tests or jaundice appear, stop therapy with methyldopa. If caused by methyldopa, the temperature and abnormalities in liver function characteristically have reverted to normal when the drug was discontinued. Methyldopa should not be reinstituted in such patients.

Rarely, a reversible reduction of the white blood cell count with primary effect on granulocytes has been seen. Reversible thrombocytopenia has occurred rarely. When used with other antihypertensive drugs, potentiation of antihypertensive effect may occur. Patients should be followed carefully to detect side reactions or unusual manifestations of drug idiosyncrasy.

Use in Pregnancy: Use of any drug in women who are or may become pregnant requires that anticipated benefits be weighed against possible risks; possibility of fetal injury can not be excluded.

Precautions: Should be used with caution in patients with history of previous liver disease or dysfunction (see Warnings). May interfere with measurement of: uric acid by the phosphotungstate method, creatinine by the alkaline picrate method, and SGOT by colorimetric methods. Since methyldopa causes fluorescence in urine samples at the same wavelengths as catecholamines, falsely high levels of urinary catecholamines may be reported. This will interfere with the diagnosis of pheochromocytoma. It is important to recognize this phenomenon before a patient with a possible pheochromocytoma is subjected to surgery. Methyldopa is not recommended for patients with pheochromocytoma. Urine exposed to air after voiding may darken because of breakdown of methyldopa or its metabolites.

Stop drug if involuntary choreoathetotic movements occur in patients with severe bilateral cerebrovascular disease. Patients may require reduced doses of anesthetics; hypotension occurring during anesthesia usually can be controlled with vasopressors. Hypertension has recurred after dialysis in patients on methyldopa because the drug is removed by this procedure.

Adverse Reactions: *Central nervous system:* Sedation, headache, asthenia or weakness, usually early and transient; dizziness, lightheadedness, symptoms of cerebrovascular insufficiency, paresthesias, parkinsonism, Bell's palsy, decreased mental acuity, involuntary choreoathetotic movements; psychic disturbances, including nightmares and reversible mild psychoses or depression.

Cardiovascular: Bradycardia, aggravation of angina pectoris. Orthostatic hypotension (decrease daily dosage). Edema (and weight gain) usually relieved by use of a diuretic. (Discontinue methyldopa if edema progresses or signs of heart failure appear.)

Gastrointestinal: Nausea, vomiting, distention, constipation, flatus, diarrhea, mild dryness of mouth, sore or "black" tongue, pancreatitis, sialadenitis.

Hepatic: Abnormal liver function tests, jaundice, liver disorders.

Hematologic: Positive Coombs test, hemolytic anemia. Leukopenia, granulocytopenia, thrombocytopenia.

Allergic: Drug-related fever, myocarditis.

Other: Nasal stuffiness, rise in BUN, breast enlargement, gynecomastia, lactation, impotence, decreased libido, dermatologic reactions including eczema and lichenoid eruptions, mild arthralgia, myalgia.

Note: Initial adult dosage should be limited to 500 mg daily when given with antihypertensives other than thiazides. Tolerance may occur, usually between second and third month of therapy; increased dosage or adding a thiazide frequently restores effective control. Patients with impaired renal function may respond to smaller doses. Syncope in older patients may be related to increased sensitivity and advanced arteriosclerotic vascular disease; this may be avoided by lower doses.

How Supplied: Tablets, containing 125 mg methyldopa each, in bottles of 100; Tablets, containing 250 mg methyldopa each, in single-unit packages of 100 and bottles of 100 and 1000; Tablets, containing 500 mg methyldopa each, in single-unit packages of 100 and bottles of 100.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486 J6AM07(707)

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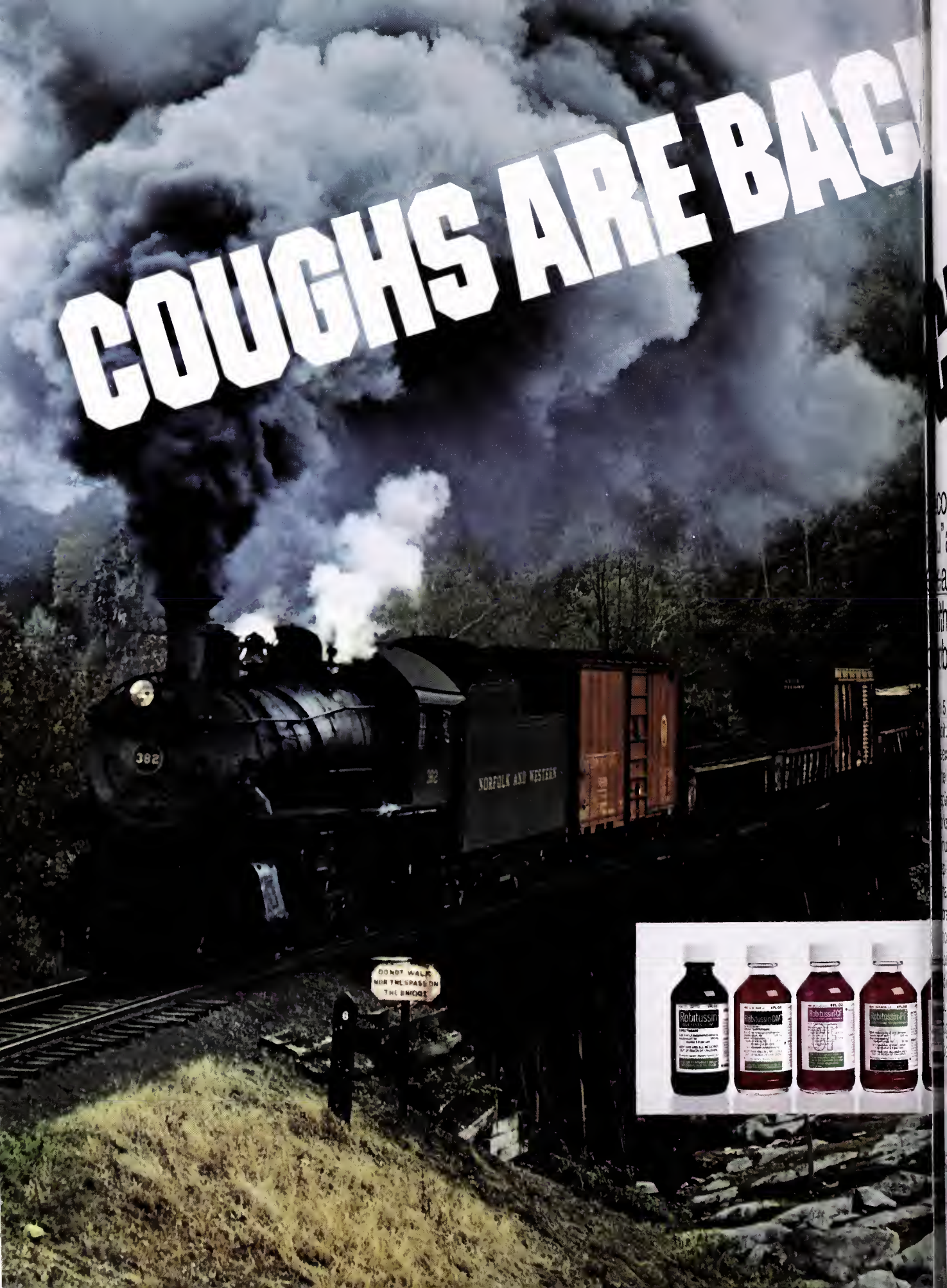
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PHOTO: Norfolk & Western Branch Train 202 west bound near Alvarado, Va. (Oct., 1956). line reaches the highest point of any railroad of the Rockies (elevation 3,577 ft.) with a imum grade of 3%. It crosses 108 bridges, e 700 ft. long! Photo by O. Winston Link.

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Hydrobromide, NF ..... 10 mg
Alcohol, 1.4%

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For many years Robins has spotlighted the expectorant action of the Robitussin cough formulations by featuring action photographs of steam engines like the one on the preceding page. In keeping with this tradition, last year the company commissioned a well-known illustrator to render full-color drawings of several classic locomotives . . . accurate to the minutest detail. Chances are you requested and received the first locomotive in this series, The William Mason, last winter. Now, the second one is available. (See below). To order your print suitable for framing, write "Robitussin Clear-Tract Engine #2" on your Rx pad and mail to "Vintage Locomotives," Dept. T4, A. H. Robins Company, 1407 Cummings Drive, Richmond, Va. 23220.

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New Year Perspective

ONE SHOULD NOT BEGIN the New Year with pessimism. Yet, everybody's frustration with inflation seems to be directed solely at the rising cost of medical care. Medical costs seem the principal target for criticism of the rising consumer price index, although medical expenses in the last five years increased about the same as everything else—39.8 per cent as compared to 38.6 per cent. From 1970 to 1975 the income of the intermediate family increased 42.1 per cent, and although social security increased 115 per cent and food went up 56.1 per cent, the major concern is the 45.7 per cent increase in medical costs[1]—and everyone ignores the significant improvement in quality of care included in this increase, small when compared to income increase. No one cares that my office rent increased 12 per cent last year and my professional liability insurance went up 63 per cent.

While brooding that medicine undeservedly is the villain in this national scenario, I came across a recent address by Marvin A. Asnes[2] entitled "The Health Care Industry in the Next Decade." The remarkable insight of this pharmaceutical executive into the health care problem deserves wide dissemination. The following excerpts briefly set forth "several key points concerning the direction in which we, both as an industry and a nation, are going."

"Point number one: Regardless of when, or even whether, National Health Insurance is enacted, the U.S. health care industry will grow at a rate significantly greater than the economy as a whole. The reasons for this statement are relatively straightforward. First, the industry has historically grown at such rates. We are already spending over 135 billion dollars a year, or 8 per cent of the Gross National Product, for health services. This year the industry will employ almost four million persons, approximately 5 per cent of the entire national work force. Depending upon whether one measures in terms of expenditures or employment, the health sector is either the country's first or second largest industry. . . . Total health expenditures have increased 7 times in the last 20 years, an annual growth rate of 11 per cent as contrasted with an increase in the GNP of less than 4 times. . . . If this historical trend continues, health care could consume over one trillion dollars in the year 2000.

"Secondly, health care is a peculiar type of service . . . it generates its own demand. In a sense, medical care needs arise because a given medical technology exists. . . . As medical knowledge expands, therefore, the health care system is able to do more for patients, and expenditures naturally increase. In addition, the more medical care that the system provides, the longer people live and the more care they consume. . . .

"Third, even though all of us in the health care system serve the public to the best of our abilities, the market structure itself guarantees growth without significant regard to cost. With over 90 per cent of the population covered by some form of health insurance, people generally do not pay directly for services and tend to seek medical care whenever they believe it is warranted. Physicians are expected by society to provide the best possible care regardless of cost. Hospitals, in turn, are motivated to provide the most modern available equipment and facilities. Furthermore, the research community, including universities and pharmaceutical and device manfacturers, is committed to rapid technological progress. All of these structural pressures drive the continued expansion of the system.

"Finally, growth of the health sector has been further stimulated by an increasing social commitment to provide universal access to quality medical care. A tradeoff obviously exists between what we, as a nation, can do and what we can afford to do. Yet, to date, whenever we have faced a choice between dollars and life, we have always opted for life. . . . HEW has estimated that by 1980, 50,000 people will receive renal dialysis services at a cost of over one billion dollars per year. It may be that we are spending too much of our health resources on too few people. We have in effect decided, however, that since the technology for treatment exists, we cannot deny it to anyone on the basis of cost."

Mr. Asnes then follows with the implications of these trends for the future of the health industry.

"The preceding rationale for health sector expansion effectively supports my initial statement that the health sector will grow regardless of the status of National

Health Insurance. Seen in this context, National Health Insurance is, in and of itself, neutral—a convenient but not essential mechanism for expressing social choice. . . .

"Point number two: The growth of health care expenditures will be more intensively monitored in the future, and the primary monitor will be the Federal Government. With so many pressures for growth, it will be essential to develop a mechanism to channel the costs of the system in a rational manner. It is inevitable that the Federal Government will have a major role in this process. . . . Please note, however, that I have carefully used the word 'monitor' rather than 'control' in describing the Federal Government's involvement. Because of the complex structure of the health industry and the enormity of the control task, the Federal Government's role will probably be that of setting standards of care, mediation and subsequent monitoring of expenditures. . . . Issues concerning "how much", "what kind" and "at what price" will be resolved in an on-going political negotiation in which the public, physicians, hospitals, and private industry bargain with each other while the Federal Government acts as mediator and monitor of the result. . . .

"Point number three: Industry growth clearly means business opportunity, but government monitoring will require a more sophisticated approach to identify and capitalize on this potential and will place an increasing emphasis on cost effectiveness and cost benefit to the system. Specific new products will have not only to provide improved effectiveness but also real and substantial cost savings. . . . Similarly, entirely new technological developments will be subject to more careful cost-benefit analyses to assure that any added costs involved will produce broad based social and/or economic benefits to the system. . . . It obviously will be in a manufacturer's best interests to anticipate and initiate such evaluations of his products and thereby become an effective participant in a government monitoring system designed to assure a generally cost-effective health care system.

"To summarize, in the 1980's, health care expenditures will continue to grow at a significant rate. It is expected that such growth will be monitored by the Federal Government. . . . Rather than a repression of private initiative, monitored growth of the health industry will continue to provide significant opportunities in an essentially free market environment but will require a re-direction of effort to capitalize on such potential. Those companies with the foresight and resources necessary to take advantage of these opportunities not only will prosper but also will contribute to the cost effectiveness of the health care system itself."

These remarks make sense. There is no way to stop the growth of the health care industry. The free enterprise system in America is not easily going to be displaced. Medicine has many and powerful allies who believe as we do. If a good cause is vigorously pursued good judgement usually prevails in our democratic system. My enthusiasm for addressing the problems is restored. Now, I am ready for the New Year.

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2 Mr. Asnes is Executive Vice-President of Becton, Dickinson, and Company. The address was presented on the occasion of the Fiftieth Anniversary of General American Investors Company, Inc. at a Special Seminar on "The Challenge of the 1980's", on November 9, 1976.

Replantation

ABOUT TEN YEARS AGO, one of the early successful replantations of an arm was performed at Colorado General Hospital. In the last few years, successful digital replantations have been performed around the world.

The advancement of microsurgical technics and organized team approach in individual centers have now made this procedure practical and worthwhile. On digits, the survival rate approaches sixty-one percent for complete digital replantations and eighty percent for incomplete. Only certain amputations should be considered for replantation. In the hand, the younger the patient, the better. The thumb, of course, is the most important digit and should receive more consideration for replantation .

Emergency room physicians and all other primary care physicians should be aware of this possibility for treatment of an amputation.

Compelling reasons to attempt replantation are (1) isolated thumb amputations proximal to the IP joint (2) multiple digital amputations (3) solitary finger amputation in a compromised hand, and (4) patients with special occupational needs.

Dr. John Boswick who is in charge of the Division of Hand Surgery at the University of Colo-

rado has a team available at all times for replantation of upper extremities. His office should be called by anyone who has a patient for whom the operation might be indicated. Dr. Clifford Snyder at the University of Utah at Salt Lake City has a similar organization.

The subject is presented in detail in this issue of the Journal on Page 371. The aim of this editorial is to point out that there are a few centers where this type of team is available, and to inform the physicians who will be seeing potential patients for replantation.

Mack L. Clayton, MD
Denver

To the Editor:

In the September-October, 1976, issue,[1] Dr. Muniz and Dr. Gervais wrote in a review article on lithium therapy that the registry of lithium babies in the United States has not shown an increase in malformations of offspring of mothers being treated with lithium carbonate during the pregnancy.

We would like to point out our observations published in Lancet[2] September, 1974. We encountered two infants who presented during the first hours of life with severe cyanosis, cardiomegaly, and a quadruple rhythm. Cardiac catherization confirmed the diagnosis of Ebstein's anomaly in both infants and both mothers had taken lithium throughout the first trimester. Lithium exposure during the two years in which these two patients were ascertained was recorded only in these two instances out of 733 teratogenic histories attained.

In reviewing the lithium registry at that time[3] nine children had been born with major malformations. What was not emphasized in the report from the register was that six of the 118 lithium babies had congenital cardiovascular diseases including ventricular septal defect, mitral atresia, coarctation of the aorta, tricuspid atresia and two cases of Ebstein's anomaly. This is a five-fold increase over the expected frequency for congenital heart disease. More striking is the recognition that three infants had tricuspid malformations, two of which were Ebstein's anomaly (which represents a 400-fold increase over the expected frequency).

With our two patients plus the six from the lithium registry, eight infants with congenital heart disease (four of whom were Ebsteins) were born following maternal exposure to lithium during the first trimester. Although lithium exposure may not be attended by the high risk of a drug such as thalidomide, our data, taken with that of the register, was strongly suggestive of an etiological relationship in susceptible individuals.

Following our initial observation and with additions to the lithium registry, Weinstein and Goldfield[4] have expressed concern over the possible teratogenicity of lithium carbonate in pregnant mothers particularly in respect to cardiovascular malformations and more specifically Ebstein's anomaly.

We felt that the readers of the Journal should know of this association.

Audrey H. Nora, MD
James J. Nora, MD
Department of Pediatrics
University of Colorado Medical Center

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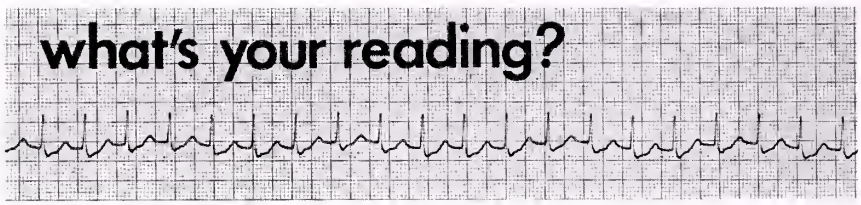
REGRETS

We regret that the November-December issue of the Rocky Mountain Medical Journal did not reach your office until the end of December. It was necessary to change printers in the middle of composition of that issue because of bankruptcy of our former printer.— Editor

OUR COVER

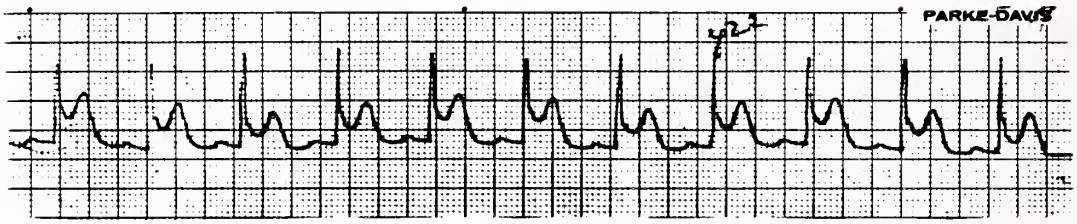
Of all Colorado dwellings that remain glorious, Georgetown's Maxwell House, whose facade is a Valentine for year-round delight, must be supreme. Marie and B.F. Potter built a simple house in 1867, but in 1890 they found themselves at the happy end of the rainbow with mining discoveries. Within a few years silver collapsed, and Frank A. Maxwell acquired the house, whose 50-year residency in it gave it its name. Thanks to Don Miller, Boulder, for still another valuable photographic memento.

what's your reading?



Prepared by Jonathan Abrams, MD, Albuquerque, New Mexico *

TELEMETRY



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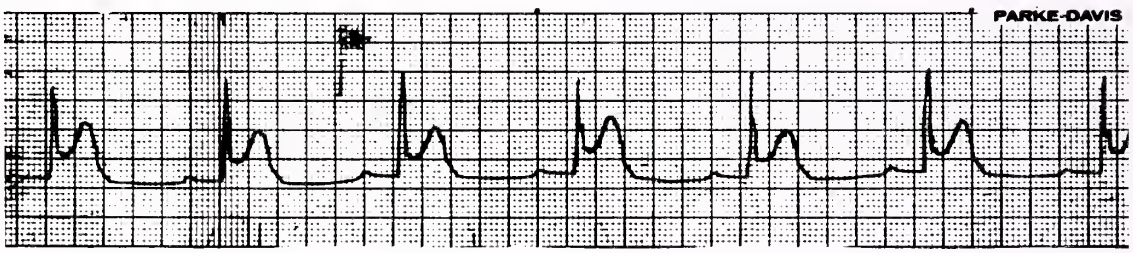

This continuous tracing was obtained from an emergency rescue squad vehicle equipped with telemetry. The patient was a 51-year-old male with chest pain, who subsequently was proved to have had an inferior wall myocardial infarction.

What is responsible for the sudden and abrupt slowing of heart rate?

See Page 28

* Dr. Abrams is Chief, Division of Cardiology, and an Associate Professor of Medicine, University of New Mexico School of Medicine, Albuquerque.

Digital replantation

Guidelines for selection and management

Ueli Buchler, MD, Dennis B. Phelps, MD, and John A. Boswick Jr., MD, Denver

"Just because an operation can be done is no reason that it ought to be done"
— Ronald Malt

Since the first microsurgical salvage of a subtotally amputated and ischemic digit in 1962[11] and a completely detached thumb in 1965,[12] successful digital replantations have been reported in over 240 instances.[3,13,15,19,20] Physicians should be aware of these developments and be able to advise their patients concerning the possibilities of replantation. Emergency room specialists and primary care physicians in particular need a working knowledge of replantation for they often initiate the treatment program and may contribute to the success of the effort.

This communication will present the expected results for digital replantation, discuss indications for this procedure, and offer guidelines for the management of these patients.

CASE REPORT

A 61-year-old white female sustained a nearly complete amputation of her left thumb on August 6, 1975, when it was caught in the fan-belt assembly of a heavy machine at work. She was sent to the University of Colorado Medical Center with her hand loosely wrapped and the thumb cooled by an ice bag, arriving 1 hour and 15 minutes after injury. There was a complete dislocation of the MP joint (Fig. 1) and all soft tissues were disrupted except the long flexor and extensor tendons (See Fig. 2). The wound was relatively clean but exhibited significant soft tissue injury.

The patient was in generally good health. She had been taking hormone replacement therapy since a thyroidectomy in 1964. The remainder of her past medical history was irrelevant.

Despite her age and the unfavorable condition of the tissues, an attempt at replantation was considered justified. Pre-operative studies were quickly performed and the patient was taken directly to the operating room which had been notified and prepared.

After an axillary block utilizing Marcaine®, the thumb was cleansed but very little debridement was performed. A tourniquet was inflated only during preparation of the stump. The operation consisted of fusing the MP joint (shortening the proximal phalanx 5 mm.) and anastomosis of the radial proper digital artery and two dorsal veins, using 10-0 nylon sutures on microvascular needles. The total ischemic time was less than four hours. A small local flap was used to cover the volar portion of the wound. A loose bulky dressing was applied and the hand placed in a slightly elevated position.

The patient was given Dextran-40, Heparin, and buffered aspirin in the immediate post-operative period. Due to swelling of the hand on the 2nd post-operative day, fasciotomy was considered. Anemia necessitated transfusion with 2000 cc of whole blood over the first four days and an additional 500 cc on the 11th. Dextran-40 was discontinued after three days, Heparin was used for two weeks in doses dictated by the daily PTT determination. Vasospasm on the 11th day responded to xylocaine infiltration about the radial artery at the wrist. A small split thickness skin graft provided complete wound closure on the 12th day of admission.

The patient was discharged on the 21st day, on buffered aspirin, 600 mg. three times daily, and her previous thyroid replacement therapy. Salicylate anticoagulation was discontinued at six weeks.

The metacarpo-phalangeal arthrodesis was solid at eight weeks. Reconstruction of the thumb web space and microscopic repair of the ulnar proper digital nerve were performed two and one-half months after replantation. While function is not normal, the patient is extremely satisfied with the final functional and cosmetic result. (Fig. 3).

From the University of Colorado Medical Center. Dr. Buchler is Fellow in Hand Surgery, supported by a grant from the Berne Research Council of the Swiss National Foundation. Dr. Phelps is Assistant Professor of Hand Surgery, and Dr. Boswick is Professor of Surgery, and Chief, Hand Service, The University of Colorado Medical Center, Denver.

Rationale

The initial aim of digital replantation is survival of the amputated part. Survival depends on numerous factors; age of patient, type and level of injury, completeness of amputation, amount of contamination, time of normothermia before cooling, time of hypothermia, operative technic and experience of the microsurgeon. Improvement in microsurgical equipment, organization of specialized replantation teams, refinement of technics, continuous training on small animals and further understandings of biologic factors responsible for failures have greatly improved the survival rate, which currently approaches 61 per cent for complete digital amputations and 80 per cent for incomplete injuries.[17]

Viability is the first but not the only aim in replantation. Prolonged hospitalization, extensive rehabilitation, the occasional necessity of subsequent procedures, and the high cost could not be justified if the functional outcome of the replanted part were inferior to the result of more common secondary reconstructive procedures. In contrast to replantation at more proximal levels of the upper extremity, which are still controversial unless performed under the most favorable conditions, digital replantations have led to a good functional recovery in most instances.

The function of the replanted digits vary, but the return of sensibility and motion is usually impressive. O'Brien [15] has reported 23 successful cases whose 2 point discrimination ranged from 2-10 millimeters and whose range of motion, though never complete, was functionally useful and allowed pinch and grasp.

There are no miracles, and a replanted digit will never be completely normal. Each structure obeys the laws governing its healing. Recovery of sensibility depends on nerve regeneration no matter how sophisticated the repair. Tendon gliding will be limited by adhesions that must form to allow revascularization and healing. There is no evidence yet that restoration of circulation after the first few hours of ischemia causes individual tissues to heal differently than injured tissues with intact circulation, except for the formation of a denser scar which in some cases may interfere with nerve and tendon healing. In these instances, secondary nerve repair and two stage tendon reconstruction can improve the final result.

Risks of replantation seen in proximal upper extremity amputations (hypovolemic shock, cardiac arrhythmia, metabolic acidosis, acute tubular necrosis and sepsis)[9,14] have not been reported in digital replantations. Gastrointestinal bleeding may occur due to anticoagulants but can be handled by their reduction or discontinuation without a greatly enhanced risk to the patency of the small vessel anastomosis. Local complications, such as massive edema, bleeding, hematoma formation, thrombosis or local infection are comparatively rare and not systemically significant. Reflex sympathetic dystrophy has not been reported. Failure of replantation with subsequent amputation does not endanger the remainder of the hand nor preclude conventional reconstruction.

Fig. 1: Roentgenogram showing complete dislocation of the MP joint of the thumb.

Digital replantation has become a well-documented, low-risk procedure which offers a reasonable chance for the functional preservation of valuable and sometimes irreplaceable tissue.

General Indications

In isolated thumb amputations, especially proximal to the distal third of the proximal phalanx, no reconstructive procedure is simpler, less expensive and functionally better than replantation, with the possible exception of pollicization where the hand is deprived of a normal finger. Replantation of an amputated thumb is a most re-

Fig. 2: Drawing showing avulsion of all tissues except the long flexor and extensor tendons.

Fig. 3: The appearance of the thumb three months after replantation.

warding microvascular procedure and should be attempted whenever possible.

In isolated index amputations, successful replantation will not produce the fine sensibility or full motion necessary for precision activities. The patient is likely to exclude it, because the long finger is a ready substitute. It is preferable in most instances to close the amputation primarily and to consider index ray amputation if the stump is not functionally reintegrated. Index replantation may be justified under special circumstances such as overwhelming desire of the patient, incompleteness of the amputation, occupations requiring all four fingers and ideal injury and time conditions.

Isolated long, ring and small finger amputations rarely interfere with the function of the hand. Replantation is justifiable when the lesion is incomplete, the occupation requires all fingers or cosmesis is an important factor.

In multiple digital injuries, however, replantation is often the only means to preserve intricacy of function. The preservation of even one digit may greatly improve funetion or allow later reconstruction. In hands with previous amputations or other disabilities, the amputated part must be carefully assessed. An already compromised hand, functioning through the use of complicated patterns of substitution, may be greatly impaired by a seemingly minor anatomical loss.

Influencing Factors

Several other factors influence the decision of whether an attempt at replantation should be made.

AGE: Digits have been successfully replanted in a 20 month old child [19] as well as in patients over 70 years of age.[17] In children, the technical feasibility will depend on the individual vessel size with a rapidly decreasing patency rate in vessels under 0.5 millimeters of external diameter. Cooperation and functional recovery in older children and young adults are in general so excellent that an attempt at replantation should be made in this age group whenever possible. A well defined upper age limit does not exist. The generally poorer return of function depends more on the biologic than the chronologic age.

OCCUPATION: Patient needs must be compared with the result which can be obtained with or without replantation. Most manual workers suffer no practical disability with solitary finger amputations and sometimes even with multiple distal digital loss. A musician or a typist, however, may be severely incapacitated by the loss of even a single digit and replantation should be considered although the result may not provide the dexterity necessary to restore those skills. The majority of patients lie between the extremes of functional demands and many of our decisions will depend on the patient's attitude. Another consideration relates to the nature of the employment: Self-employed individuals may not be able to forego the loss of time and income that the replantation program necessitates.

HAND DOMINANCE: Under most circumstances, restoration of function will be more important for the dominant hand and the patient's motivation for rehabilitation is likely to be greater.

GENERAL HEALTH: Chronic and disabling illnesses are frequently contra-indications for digital replantation. Peripheral vascular disease, including diabetes, may interfere with survival. Neurologic disorders may jeopardize the functional recovery of not only the replanted digit but also the remainder of the hand.

ASSOCIATED LESIONS: If the patient has multiple injuries, proper attention should be given to more important problems, and priorities set. Replantation can be attempted unless there are life-threatening conditions, severe injuries to more proximal portions of the involved extremity, or associated problems which exclude anticoagulation.

PSYCHOLOGIC FACTORS: The character of acutely injured patients is difficult to assess. Nevertheless, psychologic factors must be evaluated, because personality

weakness, low intelligence, or psychosis provide a poor basis for functional rehabilitation, even if replantation is successful. With insufficient motivation for restoration of function, the patient will quickly lose interest in the physical and occupational therapy program and after prolonged disability, may develop behavior disturbances.

TIME AND COOLING: Skeletal muscle deprived of its blood supply undergoes significant metabolic and patho-physiologic changes in one hour under normothermic conditions and develops histologic alterations within two hours of ischemia,[18] After six hours of normothermic anoxia, striated muscle is irreversably damaged and will develop patchy fibrosis, loss of power and amplitude, and contracture. Cooling to 4° centigrade improves tolerance to anoxia and reduces plasma sequestration which is largely responsible for the edema that leads to contracture and loss of motion. Peripheral nerve tissue is not as oxygen-dependent as muscle and in animals loses excitability after 12 hours of normothermic anoxia.

These facts are important in digital replantation. If muscle is included in the amputated part, its function will be poor unless the blood flow is restored to a cooled part within 6 hours and in an uncooled part within 3 hours. Small amounts of muscle in an amputated thumb may be excised with the advantages of less postoperative edema, lower risk of vein compression and reduced possibility of contracture. Data are not available that correlate ischemic time and functional result. Currently, uncooled digits should not be replanted beyond six to eight hours, cooled digits beyond 24 hours, and frozen digits not at all.

TYPE OF INJURY: Guillotine and moderate crush amputations are most suitable for replantation, with a survival rate of 96% in expert hands[16] and the best chance for optimal function. Avulsion and severe crush injuries, multilevel lesions, multiple fractures, soft tissue loss, or gross contamination compromise the viability and recovery and may preclude the possibility of attempting replantation.

LEVEL OF INJURY: The survival rate for amputations around the MP, PIP and DIP joints are 72, 65 and 33 percent respectively.[20] Thus, not only for functional, but technical reasons, replantations in the region of the IP joint of the thumb and distal to the middle phalanx of the fingers are rarely indicated.

COMPLETENESS: Since the results of replantation in incomplete digital amputations are so promising,[15] no opportunity should be lost to save valuable tissue. A decision must be made regarding the adequacy of the remaining blood supply. If it is not obviously sufficient and tissue viability is not certain, early microvascular repair should be performed.

FINANCIAL STATUS: The cost of replantation, hospital stay, rehabilitation and loss of income will force some patients to exclude themselves from replantation.

AVAILABILITY OF MICROSURGERY: The development of microsurgery centers is evidence of the advances in this field. Hand surgeons have found magnification and micro-technic beneficial, and thus microsurgery will undoubtedly become more widespread. Hopefully, the availability of centers will increase to the point that replantation candidates can reach them within three hours following the accident. In most centers, at least two surgeons trained in microtechnics are available at all times.

OPERATIVE FINDINGS: The final stage of the evaluation however, is the microscopic assessment of the injured tissues. Excessive damage to vessels, visible only with magnification, may make replantation impossible.

The final decision should not be anticipated by the personnel at the primary care facility but left to the replantation team, which must correlate the patient's wishes with the technical factors under consideration.

Emergency Room Care

No opportunity should be missed to instruct lay personnel to properly apply compression dressings and to save all amputated parts. Amputated tissue should be preserved in the following fashion: The part is wrapped in a sterile towel moistened with Ringer's Lactate, enclosed in a plastic bag which is placed upon ice-water (Fig. 4) and kept with the patient. Dry ice is not used. With incompletely amputated digits, it is important to place the part into the proper position and to immobilize it to protect the remaining vascularity.

The emergency physician is important to the replantation effort, since part of the success depends on appropriate psychologic approach, adequate care of the wound and the amputated part, and expedient referral. Important information regarding the exact circumstances of the accident should be obtained from witnesses who might not be available later and recorded as part of the initial history. This data includes the exact time and mechanics of the accident, the tool or machine involved, the length of time the amputated part was crushed or otherwise damaged, the time of onset of cooling, and the degree of contamination. The emergency room history and physical examination will help rule out contra-indications related to other injury or systemic illness.

Following this general appraisal, the dressing is removed, bleeding controlled by elevation and pressure, the stump and the amputated part are inspected under sterile conditions. If the criteria for replantation are fulfilled, the following steps are undertaken:

Gross contamination may be removed by irrigation with lactated Ringer's solution. Debridement is not performed, nor are any instruments

used to probe the wound or obtain hemostasis. A sterile dressing is applied to control bleeding. In an incomplete amputation, the injured part is properly aligned, immobilized and cooled. A completely amputated part is preserved as described above. The microsurgery team is notified, to obtain its advice if the indication is questionable, to ensure its availability, and to allow for preparation. While rapid transfer is being arranged, x-rays of the hand and the amputated part are obtained, tetanus prophylasis is accomplished and Dextrose 5 per cent in normal saline is started intravenously. The patient is given cephalothin, 25 mg/kg, aspirin, 10 mg/kg either PO or rectally, and sufficient pain medication. The amputated part and all available documents are kept with the patient. The patient and his family should never be told that replantation will definitely be attempted, that all attempts are successful or that normal function will ensue.

Replantation

At the replantation center, the patient is evaluated. He is informed in detail about the procedure, chance of survival of the replanted part, risks involved, possibility of subsequent procedures, rehabilitation program, cost, and expected functional result. Properly hydrated and sedated, he is taken to the already-prepared operating room and receives general or regional anesthesia. Axillary block with Marcaine® has the advantage of long-acting chemical sympathectomy with increase of blood flow. As the anesthetic is administered, the amputated part is prepared by one surgical team. This includes identification of structures, meticulous debridement and preparation of bone, vessels, nerves and tendons. Perfusion is not used. As soon as anesthesia is accomplished a second team can begin preparation of the amputation stump under tourniquet control. In multiple digit amputations, not all parts may be suitable for replantation. Those which are may be transferred onto the stumps with greatest functional importance.

The replantation itself consists of shortening of bone and appropriate internal fixation, microvascular repair of at least one artery and preferably two veins, and further primary repair of digital nerves and tendons if possible. The reader is referred to articles by Acland,,[1,2] Buncke,[4] Cobbett,[7,8] Lendvay,[13] O'Brien [15,16] and Tsai [20] for detailed descriptions of replantation and microsurgical technic.

After a loose closure, the hand is dressed in a non-constrictive bulky dressing, including a volar splint. The fingertip is left exposed for observation. The arm is moderately elevated on pillows and kept abducted at the shoulder and extended at the elbow. Adequate hydration is mandatory. Immediately postoperatively, Dextran 40 at a dose of 5 milligrams per killogram per day is used for five days.[22] Heparin at a dose determined by the result of the partial thromboplastin time is given for two weeks [21] and aspirin 10 mg/kg/twelve hours is administered and continued for four weeks. [6,23] Should bleeding occur, Heparin and then Dextran is discontinued. Sympathetic blocks, either as stellate ganglion, axillary or proximal peri-arterial blocks may be used as needed during the first several days. If the patient needs sedation, Chlorpromazine 1 to 4 milligrams/kg/day is preferred for its combined sedative and vasodilatory effects.

Monitoring includes observation of color, temperature and capillary filling every 15 to 60 minutes. Photoplethysmography and doppler flow measuring may be useful in questionable situations, but are probably not as reliable as the clinical assessment. The blood pressure is taken every two hours on the contra-lateral extremity, and the hematocrit and PTT are repeated daily.

After the first postoperative day, the wound and adjacent tissues may swell and need decompression from tight dressings or constrictive skin closure. Usually the edema subsides after formation of lymphatic anastomoses which appear after the fourth day. If signs of arterial or venous insufficiency occur, proper positioning is tried first. The pallor and coolness of arterial insufficiency may respond to a more dependent position, while venous engorgement may require greater elevation. Lack of improvement suggests either vascular spasm or a partial or complete thrombosis. Periarterial infiltration well proximal to the anastomosis with Xylocaine is attempted to rule out spasm, but if it fails (observation for a period of time is required), re-operation is necessary.

Early active motion of shoulder, elbow and all uninvolved joints as well as isometric exercises and ambulation are started on the first postoperative day. At approximately two weeks the patient may leave the hospital, depending upon his progress and cooperation. After three weeks, the external immobilization of the involved digit is discontinued and further rehabilitation started. Secondary procedures, if necessary, are considered after six weeks.

Summary

Replantation of amputated digits has become an accepted surgical procedure. The advancements in microsurgical technic make successful replantation possible in over 60 per cent of cases. In justified attempts the functional outcome is usually good.

This report identifies factors which influence the success of any individual attempt, and outlines the indications and contraindications for digital replantation. Compelling reasons to attempt replantation include (1) Isolated thumb amputations proximal to the IP joint, (2) Multiple digital amputations (3) Solitary finger amputations in previously comprised hands or patients with special occupational needs or expressing overwhelming desire for replantation.

The management of the potentially replantable amputation is also presented. For such an effort to be successful, care must begin at the place of the injury, continue in a precise and systematic manner at the emergency or primary care facility and culminate at the microsurgical center with the utilization of exacting technics and meticulous postoperative management.

Replantation is an involved process, requiring pre-operative judgement, technical skill, and diligent post-operative management. It cannot be undertaken by the casual observer or occasional microsurgeon with the hope of success.

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Transvenous pacing in a premature infant

Reversal of heart block and congestive failure

Owen P. O'Meara, MD, Philip P. Oliva, M.D., Alan M. Lake, MD, John C. Breckinridge, MD, Richard S. Buswell, MD, Eugene S. Lapin, MD, Denver

TEMPORARY TRANSVENOUS PACING has been performed in full term infants,[1-2] but has not been described in a premature child. The purpose of this report is to illustrate use of transvenous pacing in an 1150 gram infant. Of additional interest is the demonstration of an increased stroke volume and a normal to increased ejection fraction during congestive heart failure, and reversal of the heart failure by increasing the heart rate from 60 to 130 per minute.

CASE REPORTS

A 990 gm female infant was the product of a 30 week gestation of a 24 year old woman. Her Apgar rating was 1 at 5 minutes and she was intubated in the delivery room. On admission to the nursery her heart rate was 140 per minute, cardiovascular examination was normal, and chest X-ray was normal. She was extubated within the first day. At 14 days she developed toxic necrotizing enterocolitis which resolved after seven days of ampicillin (200 mg/kg/day) and gentamicin (7.5 mg/kg/day) therapy.

At 23 days she developed a sinus tachycardia, tachypnea, hepatomegaly and increased heart size by radiograph. She was digitalized with a total of 50 micrograms of intramuscular digoxin, based on a total digitalizing dose of 50 micrograms per kilogram (weight 1.2 kilograms). Thirty micrograms were given initially followed by two doses of 15 micrograms each 12 and 24 hours later. One maintenance dose of 5 micrograms was given the following day. At that point the heart rate fell to 60 per minute and the electrocardiogram showed 2:1 A-V block (Fig. 1A) with an occasional Wenckebach period. Digoxin was discontinued and atropine was given on several occasions without benefit. The next day she developed complete heart block with a junctional pacemaker (narrow, unchanged QRS complexes at a rate of 60 per minute) and developed evidence of increasing failure with a loud S_3, pulmonary rales, hepatomegaly, decreased urine output, decreased arterial p02 and further cardiac enlargement. Isuprel was administered by intravenous infusion up to a dose of 0.7 ugm/kg/minute with a consequent increase of heart to 80 and a modest improvement of the heart failure. A digoxin level on day 33 (8 days after the digoxin was discontinued) was 2.5 ngm/ml. A second digoxin level on day 37 was zero.

On day 38 cardiac catheterization was performed through a cutdown on the right saphenous bulb. All pressures were normal and there was no evidence of a shunt. Complete heart block was present and the ventricular rate was 96 per minute. A left ventricular cineangiogram showed a normally situated left ventricle giving rise to a normal aorta. The ventricular cavity was large but contraction appeared vigorous. The end-diastolic volume was 58cc/m^2, the end-systolic volume 9.5cc/m^2 and the stroke volume 48.5cc/m^2. The end-diastolic volume and stroke volume were increased.[4] The systolic ejection fraction was calculated to be 83%. A#4 bipolar pacing catheter was placed in the right ventricular outflow tract (figure 2A and B) and pacing was instituted at a rate of 130 per minute.

Following the institution of pacing, signs of failure quickly resolved with loss of the gallop, hepatomegaly and rales. Urine output and oral intake improved. Arterial pO^2 increased significantly, and heart size became smaller but not normal. On day 40 the pacing electrode withdrew to the right atrium where atrial pacing was achieved with 2:1 A-V conduction to a ventricular rate of 80 per minute. Mild failure returned which was easily controlled by furosemide. On day 41 steroids were begun and by the next day atrial pacing at a rate of 100 per minute resulted in 1:1 A-V conduction. By day 42 she was in sinus rhythm with only brief periods of Mobitz I block and fixed 2:1 block. On day 44 her rate was 120

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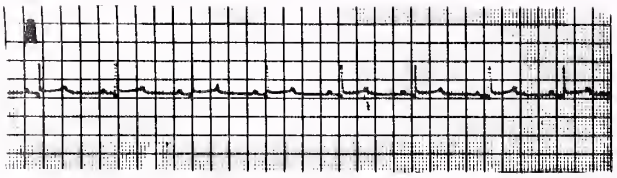
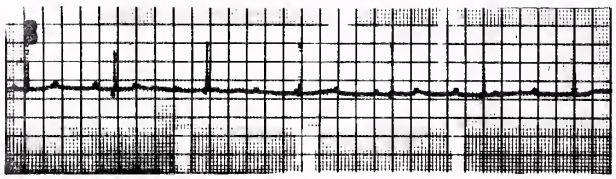
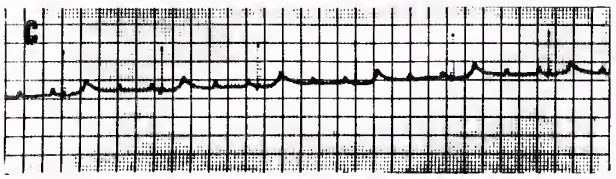


Fig. 1 (A). Day 25. Rhythm strip demonstrating 2:1 A-V block. Atrial rate 146 per minute, ventricular rate 73 per minute.



(B). Day 26. Complete heart block with narrow QRS complexes. Ventricular rate 60 per minute.



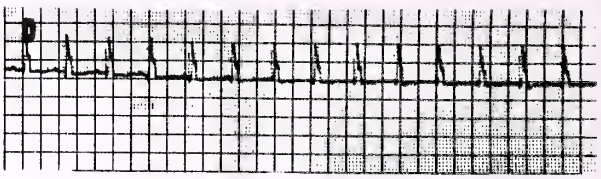
(C). Day 32. 3:1 A-V block. Atrial rate 166 per minute, ventricular rate 56 per minute.

per minute with 1:1 A-V conduction and brief periods of Mobitz I block. The pacing wire was removed without incident on that day. She continued to do well and the steroids were discontinued, on day 48. The heart rate reached 160 per minute by day 50 and remained normal thereafter. First degree A-V degree A-V block has been consistently present since sinus rhythm was restored. Fig. 1 illustrates sequential rhythm strips showing various degrees of A-V block prior to pacing and the subsequent resolution of the conduction defect.

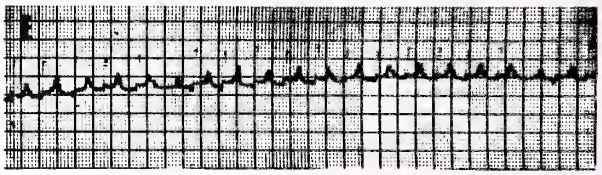
Viral cultures of the nose, stool and urine performed on day 31 and day 40 were negative.

Discussion

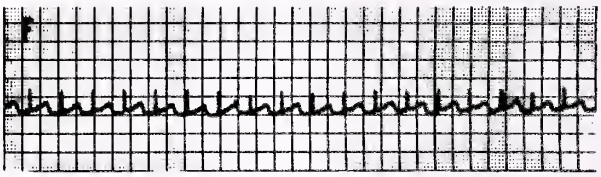
This child weighed 1150 grams and was 38-days-old at the time the temporary transvenous pacemaker was inserted. Despite her small size no problems were encountered during six days of transvenous pacing except for spontaneous withdrawal of the pacemaker catheter from the right ventricle to the right artium. This probably occurred because the tip of the catheter had to be positioned in the right ventricular outflow tract. This site does not provide the stability of the right ventricular apex. It might be possible to obtain pacing from the right ventricular apex in an infant if the bend in the tip of the catheter were appropriately modified for an infant's heart. No immediate or subsequent problem with venous insufficiency of the leg occurred.



(D). Day 39. Right ventricular pacing at rate of 130 per minute. Independent atrial activity is present.



(E). Day 53. Sinus tachycardia at rate of 170 per minute with two blocked atrial impulses. A Mobitz type I mechanism is probable. The increasing PR interval prior to the blocked P wave is difficult to measure at this heart rate and paper speed, but the PR interval following the blocked P wave is shorter than the preceding ones.



(F). Day 71. Sinus tachycardia at rate of 166 per minute with persistent first degree A-V block.

At the time the child was catheterized she was in congestive heart failure. Stroke volume was increased and the ejection fraction was normal to increased. Despite this compensation for the bradycardia, heart failure occurred and was reversed by

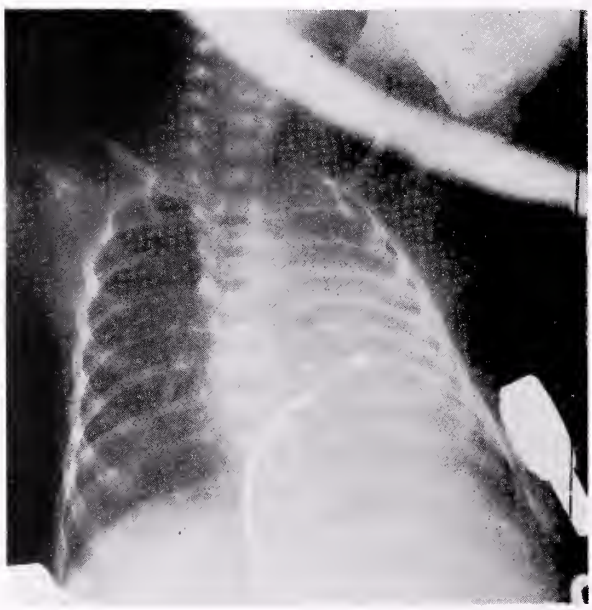


Fig. 2. AP chest film demonstrating cardiomegaly and the position of the bipolar pacing catheter.

pacing. Rapid resolution of heart failure during pacing has been seen in some full-term infants with heart block.[2,3] However, the relationship of heart failure associated with slow ventricular rates to end-diastolic ventricular volume and ejection fraction has not been established.[5] The ventricular volume measurements recorded in this infant indicate that heart failure can occur from bradycardia without concomitant left ventricular dysfunction or underlying congenital heart disease.

The etiology of the A-V conduction problem remains unknown. The initial serum digoxin level seems high one week after discontinuation of the drug. However, digoxin intoxication seems unlikely in view of the length of time that the conduction defect persisted after the digoxin was discontinued and the second serum digoxin level of zero. A viral myocarditis also seems unlikely in view of the negative cultures and the above normal stroke volume and ejection fraction. Corrected transposition and other congenital heart lesions are excluded by the catheterization information. The possible role of necrotizing enterocolitis and the antibiotic therapy are not known.

The normal QRS configuration, the ventricular rate of 60 during complete heart block and the preceding Mobitz I A-V block indicate that the A-V node or His bundle was the site of the conduction defect. Thus, this child had complete block within the A-V node or His bundle without evidence of more distal block and without ventricular dysfunction at the time of catheterization. The conduction defect was acute and reversible. The cause remains uncertain.

We would like to thank Mary Ann Fisher for her assistance in the preparation of this manuscript.

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Pediatricians' perceptions

Competence of child health associates

Claibourne I. Dungy, MD, MPH, and Henry K. Silver, MD, Denver

Pediatricians who employ or supervise child health associates perceive them to be highly skilled health professionals. The majority of pediatricians rate the ability of child health associates to assess and manage ambulatory patients to be better than that of most health workers caring for children and as good as pediatricians entering practice.

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The ultimate acceptance and utilization of new categories of health professionals may depend more on the perceptions that physicians have of the capabilities of these health workers than upon their proven abilities to deliver high quality health care and services. This report presents pediatricians' ratings of the clinical competence of child health associates in comparison to other categories of primary care providers.

Methods

Twenty-three pediatricians who employed or supervised child health associates were asked to compare the ability of child health associates to assess and manage pediatric patients in ambulatory settings with that of pediatric nurse practitioners, fourth-year medical students, "straight" interns in pediatrics (PL-1), second-

TABLE 1

Practicing pediatricians' perceptions of child health associates and other health professionals

Skill of child health associates in assessing and managing ambulatory patients compared to:	Percent of pediatricians who indicated a child health associate is:	
	As good as:	Better than:
A. Pediatric nurse practitioners	100%	100%
B. Fourth year medical students	100%	93%
C. General practitioners entering practice	100%	40%
D. "Straight" interns (PL-1) in pediatrics	92%	54%
E. Second year residents (PL-3) in pediatrics	50%	29%
F. Pediatricians entering practice	57%	0%

year residents in pediatrics (PL-3), and pediatricians and family practitioners starting in practice. Pediatricians also compared child health associates to an "average physician" in their ability to accomplish the following: (1) obtain an accurate history from children and adolescents, (2) perform a thorough physical examination, (3) obtain pertinent information of psychosocial problems, (4) synthesize the history and physical examination in order to derive the proper diagnoses, (5) formulate an appropriate plan of action based on findings from the history and physical examination, (6) know and use information about community resources, (7) recognize and appropriately refer diagnostic and management problems, and (8) provide health education, support, and counseling to parents and patients.

Child health associates had been in practice for 5 to 29 months at the time this study was carried out.

Findings

Sixteen of the 23 pediatricians (69%) completed the questionnaire The skill of child health associates in assessing and managing ambulatory pediatric patients was perceived by all of the pediatricians to be better than pediatric nurse practitioners and fourth-year medical students, and at least as good as that of general practitioners. (Table 1). Over ninety per cent perceived child health associates to be at least as good as "straight" interns in pediatrics (PL-1), while one-half of the pediatricians felt that the child health associates' abilities were as good as those of second-year pediatric residents (PL-3) or pediatricians starting practice.

Most practicing pediatricians who responded to the questionnaires reported that child health associates could perform all of the specified functions and activities at least as well as an "average physician." (Table 2). More than 85 percent of the pediatricians indicated that child health associate graduates were as good as or better than an "average physician" in obtaining an accurate history, performing a thorough physical examination, making a

TABLE 2

Pediatricians' perception of the clinical skills of child health associates in comparison with an "average physician"

Clinical Skill	Percent of pediatricians indicating a child health associate is as good as or better than "average physician"
A. Obtaining accurate history	94%
B. Performing thorough physical examination	90%
C. Obtaining pertinent information of psychosocial problems	93%
D. Synthesizing history and physical examination to derive proper diagnoses	73%
E. Formulating appropriate plan of action based on history and physical examination	67%
F. Knowing and utilizing information about community resources	88%
G. Recognizing and appropriately referring diagnostic and management problems	86%
H. Providing parent and patient education, support and counseling	100%

pertinent psychosocial assessment, recognizing and appropriately referring diagnostic or management problems, and providing patient and parent education, support, and counseling. Three-fourths of the pediatricians noted that child health associates were at least as good as an "average physician" in synthesizing histories and physical examinations in order to derive proper diagnoses, and two thirds indicated that child health associates could formulate an appropriate plan of action based on findings from history and physical examination that was as good as the physicians'.

Comment

Since physicians play a major role in deciding how non-physician health workers will function in the health care delivery system, the physicians' perceptions of the ability of new health professionals as providers of primary health care is an issue of critical importance. Physicians must be confident that new health personnel such as child health associates are competent providers of patient care before the physicians can safetly delegate to them the performance of a wide variety of clinical functions and activities.

The results of this study indicate that most pedi-

atricians who employ or supervise child health associates perceive them to be highly skilled health professionals with diagnostic and therapeutic skills in ambulatory pediatrics better than that of pediatric nurse practitioners and at least as good as general practitioners starting practice. The ability of child health associates to assess and manage ambulatory patients is perceived by pediatricians to approach that of residents in pediatrics and pediatricians entering practice.

Other studies of child health associates have demonstrated their ability to care for a large per cent of pediatric patients and provide a wide range of diagnostic, preventive, and therapeutic services to children.[1,2] Working principally in ambulatory settings as colleagues and associates of physicians,[3,4] child health associates have the knowledge and the skill to care for most of the patients seen in a typical pediatric practice [5] and, as a result have the potential of being significant providers of primary health care to children.

Before new health professionals can be utilized effectively as providers of primary care, evidence of (1) clinical competence, (2) consumer acceptance, and (3) physician acceptance must be met. Clinical competence [6,7] and consumer acceptance [8] of child health associates have previously been demonstrated. This study demonstrates that child health associates are highly regarded by employing and supervising pediatricians who consider them to be as good as most physicians in delivering ambulatory care to children and adolescents.

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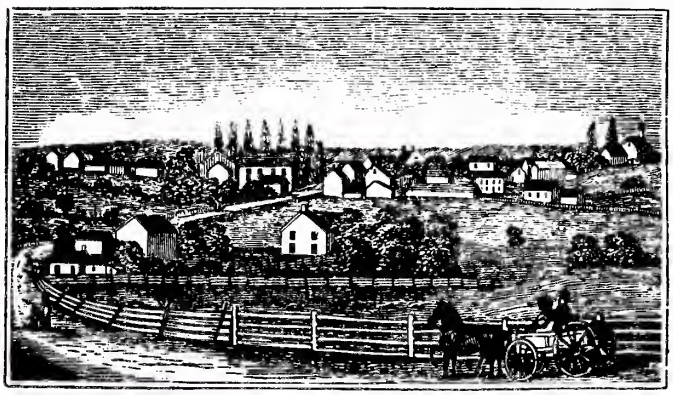

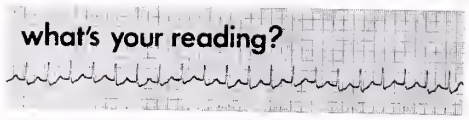

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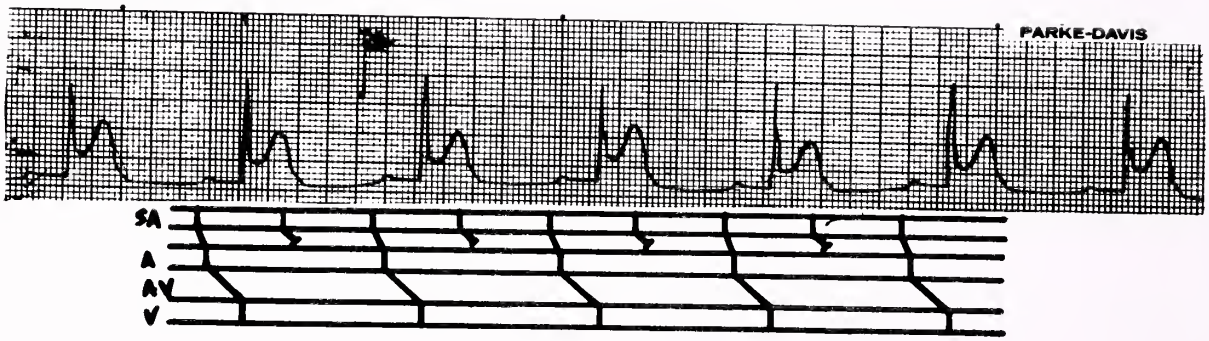
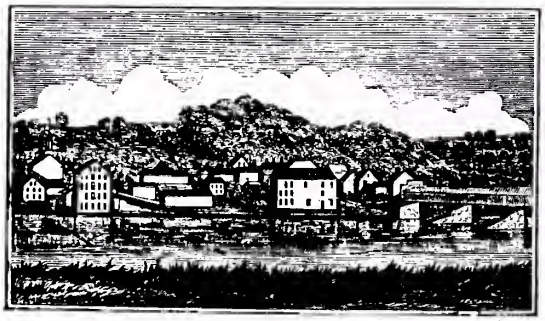


Answer:

The appearance of a sudden slowing of the sinus rate, along with an increase in the P-R interval, suggests a high level of vagal discharge which is frequently seen in inferior myocardial infarction. The mechanism may well be simple sinus slowing with sinus bradycardia and first degree A-V block. However, the atrial rate in the lower panel is almost precisely half that in the top panel. This suggests an alternative mechanism that is, *2:1 sinoatrial block.* This is diagrammed below (Fig. 3-B). The intrinsic sinus firing rate is almost identical to the faster ventricular rate (118 vs. 110 beats/min.) but every other sinus impulse fails to "get out" of the sino-atrial perinodal tissue to activate the atria and produce a P-wave; thus the effective atrial and ventricular rate is halved (to 59/min.). S-A block can only be diagnosed when there are sudden alternations in cycle length, with the longer P-P intervals representing multiples of the shortest P-P intervals (e.g. 2:1, 3:1). This phenomenon is seen in sick sinus node syndrome as well as in vagotonic states. This electrocardiographic study is a good example of the fact that conduction disturbances resulting in 2:1 block, or other multiples, can be found in any of the specialized conducting tissues of the heart, not just the A-V node.



Use of drugs for anxiety

David U. Caster, MD, Colorado Springs *

The physician must understand both the drugs and his motives for using them

Anxiety is a subjective feeling of discomfort which encompasses feelings of dread, fear, apprehension, and/or irritability, emotions usually associated with signs of autonomic overactivity such as excessive perspiration, tachycardia, palpitation, and hyperventilation, and which may or may not have a known external source. Whether we call it tension, nervousness, uptightness, or anxiety, this unpleasant subjective discomfort is, like depression, a universal emotional experience shared at times by each of us, patient or physician. It may be this commonality which has led the physician to strive with such effort to abolish these dysphoric feelings by using drugs of every description. The use of prescription anti-anxiety agents is increasing at an alarming rate with one in ten Americans taking these drugs during any three month period, at a yearly cost greater than two hundred million dollars.[1] Since general practitioners prescribe seventy-five per cent of these drugs (non-psychiatrist specialists twenty per cent, and psychiatrists five per cent), there is a need to understand why they are so frequently used in general medicine.

It is proposed that the origin of much of the excessive use of anti-anxiety agents in general medicine may result from the interaction of the physician's anxiety with that of the patient's. This occurs as a result of the role assignments which our culture places on both patient and physician and the expectations which arise from these roles. As with most of our expectations, there is no requirement that they should be rational or consciously understood. First, let us look at the patient considering the expectations he places both on himself and his physician. In our production-oriented society a person who is seen as being ill is allowed to abdicate to a greater or lesser extent from society's demands for self-reliance and productivity, and he may assume a role of greater dependency than his healthy contemporaries are allowed. While this "side benefit" of illness may be consciously recognized both by the patient and the physician, loss of productivity, increased helplessness, and ensuing lowering of self-esteem nonetheless compound the pain of disease. The role of the physician interdigitates with this concern over dependency since he is supposed to be a caring minister to those who cannot care for themselves. However, in the physician there is not only the socially desirable need for personal productivity but also the charge to heal and make others productive and independent.

What happens when the patient does not improve, or despite observable improvement feels disconsolate? One certain result is dissatisfaction by both patient and doctor, since this hopeless mood opposes the expectations of both. This failure becomes an additional source of anxiety and depression for both and may result in the physician's attempting to at least ameliorate discomfort by prescribing for this anxiety, at times in a potentially destructive manner. Treatment of anxiety is particularly problematic when the patient's complaints are undefinable, and his illness is not subject to diagnosis, since this leaves the physician without the comfort of saying, "Well, the diagnosis isn't treatable; the least I can do is help the patient face his illness." The frustrations of the physician faced by a patient with an undefinable or untreatable problem, clearly in pain,

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irritable and demanding, may result in his treating this patient from a combination of concern plus his own anxiety and hostility. Unless physicians keep clearly in mind the frequent lack of realism in some of the expectations we place on ourselves, and unless we keep a clear view of the hostility engendered in us by non-responsive patients, our own emotional involvement will complicate their treatment.

Antianxiety Agents

The antianxiety agents are frequently called minor tranquilizers, a misnomer because of its implication that there are major tranquilizers—a term which usually refers to phenothiazine-like drugs. The so-called major tranquilizers are antipsychotic drugs, which tranqualize only in the context of improving severely disturbed patients and may actually increase anxiety in the normal individual. There also needs to be a differentiation between antianxiety agents and the sedative hypnotics. Hollister [2] contends that separating drugs considered specific for anxiety (the benzodiazopines) from other sedative-hypnotics (the barbiturates, meprobamate, Placidyl, Doriden) is unjustified pharmacologically. There are, however, good series [3] which indicate not only relatively specific pharmacologic effects, which can only be reproduced with the sedative hypnotic group at near toxic levels, but also clinical superiority in treating anxiety. Possibly more important, with the benzodiazopines, there is no evidence at present of suicidal potential (unless taken in combination with other drugs, usually alcohol) and less evidence of abuse potential. Certainly, clinicians involved with drug abuse treatment see many patients who develop psychologic and occasionally physical dependency on the benzodiazopine drugs and they might caution against its abuse potential. Swanson et al.[4] have documented cases of clinical abuse which seem clearly to require collaboration between the chronically ill patient and the prescribing physician. However, there is fairly universal agreement that there is less abuse potential with the benzodiazopines than with the sedative hypnotic group. If the assumption of equal potential for controlling anxiety with that of other sedative hypnotics is made, then the lower abuse rate and lack of lethality are grounds enough for separating these agents from the barbiturates, meprobamate, glutethamide (Doriden), and the other drugs which are considerably more lethal. One of these agents, tybamate, a glycerol derivative similar to meprobamate, may be an exception in that it too may have both low abuse potential and no lethality.[5] Table 1 makes several comparisons among various psychoactive drugs.

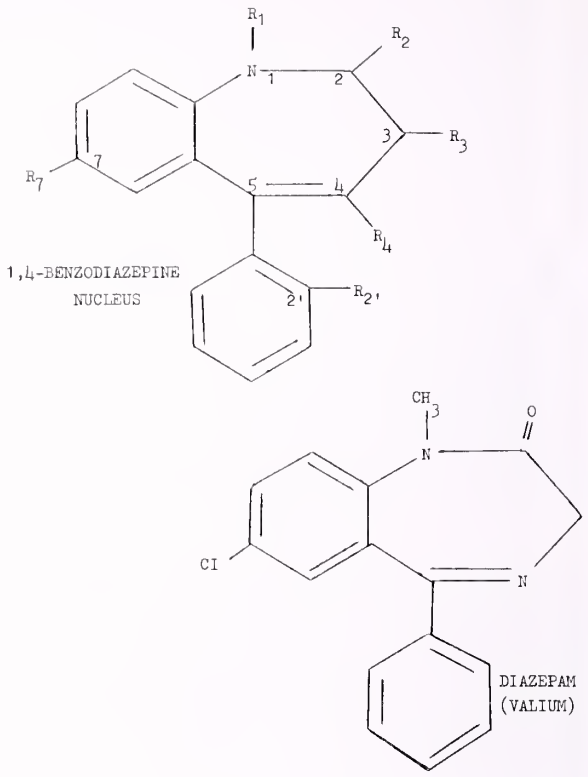

Fig. 1

The benzodiazopine nucleus (see Fig. 1) is the basic molecule, with various drugs having a variety of moieties added at the various sites. Diazepam, as an example, is shown in Fig. 1. These drugs have the following specific affects: (1) anxiolytic, (2) anticonvulsant, and (3) muscle relaxant. The antianxiety effects of these drugs have been shown in animals to stem from decreased response to nerve stimulation in the amygdaloid-hyppocampal-hypothalamic pathways. EEG electrodes implanted in the amygdaloid body show decrease in electrical activity. Benzodiazopines are considered to function as limbic system sedatives.

The anticonvulsant activities of these compounds function through an unknown mode of action. These drugs do not suppress seizures originating in specific foci, but propagation from specific foci may be suppressed and idiopathic activity is controlled. Diazepam (Valium) is considered the treatment of choice for status epilep-

TABLE 1

Pharmacological Properties of Antianxiety Drugs

	Phenobarbital	Meprobamate	Benzodiazepines	Phenothiazines	Tricyclics
Antianxiety/sedative ratio	Slight	Moderate	Moderate	Slight	Slight
Muscle relaxation	Minimal	Moderate	Great	Opposite effect	None
Anticonvulsant action	Great	Moderate	Great	Opposite effect	Opposite effect
Duration of action	Great	Slight	Great	Moderate	Moderate
Tolerance	Moderate	Great	Slight	None	None
Habituation	Minimal	Great	Minimal	None	None
Physical dependence	Slight	Great	Slight	None	None
Disturbed sleep pattern	Moderate	Moderate	None	Moderate	Moderate
Potential suicide use	Moderate	Great	None	None	Great

Modified from: Hollister, L. E., Use of Psychotherapeutic Drugs, Annals of Internal Medicine, 1973, 79:90

ticus. The muscle relaxant properties are present in all benzodiazopines but vary greatly in different members of the group. Mode of action of these drugs is unknown; however, it is known that injection into the intraventricular space causes relaxation in animals so long as the spinal cord is intact. These drugs also act directly on the nerve end-plate muscle preparation.

Since the focus of this paper is the antianxiety properties of these drugs, we will look only at specific behavioral effects which help to understand this action. Animal studies have shown that collectively the effect can cause a temporary loss of inhibition. This involves (1) increased exploratory activity, (2) decrease in active (punishment motivated) avoidance, and (3) alteration of behavioral and biochemical changes (towards the resting state) usually associated with stress. Other drugs of the sedative hypnotie group accomplish the same effects, but they do so at near toxic rather than at therapeutic doses. No dosage of tricyclic antidepressants or phenothiazines has such an effect. Assuming behavioral studies in animals can be related to the patient, the physician must understand that stress originating from reality issues and from subconscious psychologic problems can be suppressed, but the origins of the anxiety are not in either case modified by this treatment. In fact, a direct relation of the effects on behavior in animals to man conceivably can be deleterious. Patients suffering from physical or psychologic chronic illness may sustain harm if the anxiety which motivates them to develop alternate methods of dealing with painful situations in their lives should be pharmacalogically removed.

The Individual Drugs

Table 2 lists the benzodiazopine drugs available in the United States. While all benzodiazopines are fairly similar in their effectiveness against anxiety, several differences do exist. These drugs with the exception of oxazepam have long duration of action requiring twice-a-day or daily administration. Oxazepam has a short half-life with no active metabolic products, and therefore needs more frequent prescribing. Chlordiazepoxide is more reliably absorbed by injection than diazepam, however, diazepam has an intravenous form usable both for controlling seizures and extreme agitation. There is some evidence that both chlordiazepoxide and diazepam may cause episodic outbursts of aggressive behavior. While this has not been shown to occur with oxazepam, all

TABLE 2

The Benzodiazepines

Generic Name	Trade Name	Usual Daily Dose Range
Antianxiety agents:		
Chlordiazepoxide	Librium	15 to 100
Diazepam	Valium	6 to 40
Oxazepam	Serax	30 to 120
Clorazepate	Tranxene	11.75 to 60.0
Hypnotics:		
Flurazepam	Dalmane	15 to 30

these drugs may produce increasing irritability in certain patients.

Sleep studies indicate relatively little effect on rapid eye movement (REM) sleep as opposed to the effects of the sedative hypnotic group. Therefore, there is no REM rebound with its disturbing dreams such as frequently occurs with the sedative-hypnotics. This has led to the growing popularity of flurazepam as the hypnotic of choice. The benzodiazopines do have an effect of diminishing stage four sleep, and they have been used successfully in treating night terrors.

None of the benzodiazopines have been shown to have specific antidepressant effects. There has been moderate success in treating mixed anxiety and depression; some anxious schizophrenics have benefited from these drugs.

Indications for Treatment

Anxiety may be differentiated into four distinct categories: (1) anxiety neurosis, (2) situational anxiety, (3) anxiety as aspect of disease, and (4) psychophysiologic disorders. Anxiety neurosis is definable as recurring emotional state without recognizable etiology, beginning before middle age which is manifested by apprehension, fearfulness, or a sense of impending doom, and is associated with autonomic nervous system discomfort. Situational anxiety can occur at any age and it is identified in an individual of relatively normal emotional background bewildered by a specific external stress. Anxiety as an aspect of disease is a specific example of situational anxiety where dysphoric feeling is related to consequences of the illness. Psychophysiologic disorders are here considered as any condition in which emotional factors, particularly stress, have either an etiologic or perpetuating factor for peptic ulcer disease, for example.

The use of antianxiety agents in these four different groups of disorders will vary depending upon the specific condition as well as such other external events as the availability of alternate treatment. The use of antianxiety agents in patients suffering from anxiety neurosis is considered acceptable (1) when its use is intermittent and (2) for severe exacerbations. It is not the treatment of choice in those who could benefit from therapy leading to behavioral change. The use of antianxiety agents in those suffering from stress is indicated only when the anxiety interferes with successful management of stress situations. The use of antianxiety agents in the treatment of stress related diseases is a complex issue depending in some degree on what supportive psychotherapeutic measures are available and whether the illness is acute or chronic. Despite a lack of evidence for the development of dependency when patients are treated in a controlled situation, chronic use of antianxiety agents may lead to decreased drug effectiveness. The psychophysiologic disorder may be the physician's biggest problem, as drug use to control anxiety may ease symptomatic expressions of the patient's disease. However, since most of these disorders tend to be chronic, reliance on drug therapy prevents the patient's realistic dealing with changes in his living situation which would lead to removal or lessening of anxiety. Collaboration between the primary physician and a consultant psychiatrist may help clarify these difficult problems and improve the patient's treatment.

Conclusion

The antianxiety agents--specifically the benzodiazopines--are effective, relatively safe drugs when used appropriately. Their effectiveness is greatest in the hands of a physician not intimidated by the patient's poor response to overall treatment, and who limits the use of these drugs to short-lived and severe circumstances. It is inappropriate to use these drugs without assisting the patient to understand and deal more effectively with those life events perpetrating anxiey.

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Postpartum massive lower gastrointestinal bleeding

Paul Wexler, MD, Denver *

Massive lower gastrointestinal bleeding as a consequence of vaginal delivery is not reported in the modern literature. Two cases of such hemorrhage are reported. Knowledge of this entity allowed a more conservative surgical approach in the second case.

CASE REPORTS

Case 1: A 21-year-old female, Gravida 1, Para O, whose expected date of confinement was 3 April 1967, was admitted to the hospital in early labor at 41 weeks gestation. Prenatal course was complicated only by E. coli pyelonephritis at 16 weeks gestation which responded to appropriate antibiotic therapy. After a labor of 14 and 44/60 hours, the patient was delivered by low midforceps of a 7 lb. 4 oz. male infant (Apgar 7-9), over a right mediolateral episiotomy without extension. Her postpartum course was uneventful and she was discharged on her fourth postpartum day.

Twenty days postpartum the patient was seen in the Emergency Room for heavy lower gastrointestinal bleeding. Sigmoidoscopy was performed. Bloody stool was visualized to 25 cm. and no lesions were seen. The patient was admitted, a barium enema done, and sigmoidoscopy repeated. No lesions could be found. The patient's hematocrit was 39% on admission, and 24 hours later it was 29%. She was transfused with two units of whole blood. Repeated hematocrit was 33%.

Approximately 55 hours after admission massive lower gastrointestinal bleeding recurred. She was transfused with a third unit of whole blood and taken to surgery. Exploratory laparotomy was performed. Examination of the large and small bowel, stomach, duodenum, and gall bladder failed to reveal any pathology. A sigmoidotomy was done and sterile sigmoidoscope introduced. The proximal colon showed no bleeding sites or lesion. The distal colon contained fresh and old blood without any lesion identified. The scope was removed and the sigmoidotomy incision closed. The adbomen was then closed.

The patient was then placed in the lithotomy position and sterile vaginal examination and speculum examination perfromed. No pathology was demonstrated. The episiotomy was intact and did not communicate with the rectum.

Sigmoidoscopy was again performed with no evidence of lesion or bleeding site. Upon removal of the sigmoidoscope, bright red bleeding occurred. An arterial bleeder was identified at 12 o'clock at the pectinate line. This was ligated with a 3-0 chromic catgut and no further bleeding was noted. A fourth unit of whole blood was transfused and the hospital course was subsequently unremarkable.

Case 2: A 28-year-old female, Gravida 2, Para 1, whose expected date of confinement was mid January 1974, was admitted to the hospital for elective induction of labor on January 24, 1974. The cervix was 3-4 cm. dilated. An intravenous pitocin drip was begun. After a labor of 3 and 01/60 hours, a 6 lb. 2 oz. female infant (Apgar 9-10), was delivered by low forceps over a midline episiotomy without extension. The patient's postpartum course was uneventful and she was discharged on the third postpartum day.

The patient was readmitted on the fifth postpartum day with massive lower gastrointestinal bleeding. Sterile vaginal examination and speculum examination showed no fresh bleeding. Sigmoidoscopy showed bloody stool to 25 cm. No bleeding site or lesion was identified. Admission hematocrit was 32%. Twenty-four hours post admission the hematocrit was 27%. Bed rest was prescribed for 24 hours then progressive ambulation permitted.

Approximately 48 hours after admission, massive lower gastrointestinal bleeding recurred. She was transfused with two units of whole blood and taken to surgery. Direct visulation of the anal area revealed an arterial bleeder at 12 o'clock at the pectinate line. This was easily controlled with a single suture of 3-0 chromic catgut. No further bleeding was noted. The episiotomy was intact and did not communicate with the rectum. The remainder of the hospital course was unremarkable.

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Comment

Since the bleeding site was identified at the pectinate line in both cases, these instances of massive hemorrhage might be more properly described as anal. It might be conjectured that a laceration, presumably of one of the hemorrhoidal arteries, was initially concealed by edema, or that trauma from delivery caused arterial thrombosis, delayed arterial wall breakdown, and subsequent hemorrhage. Voluntary external rectal sphincter spasm might explain the reflux of blood into the distal colon. Both deliveries were by forceps with episiotomy. Neither episiotomy, however, communicated with the anal-rectal area.

The massive lower gastrointestinal bleeding reported in this paper is the first written report of which we are aware. It seems probable that other unreported cases have been encountered. By awareness of the condition, major surgical intervention was avoided in the second case.

Glutethamide poisoning

N. G. Orfanakis, MD, and W. B. Galloway, MD, Denver

A poor correlation exists between blood levels of glutethamide and the clinical state. Ambre and Fischer reported a patient to support their hypothesis that 4Hg (4-hydroxy-2-ethyl-2-phenylglutarimide) a metabolite of the drug, is more important as a cause of coma than glutethamide itself.[1,2] Recently we studied a patient who supports their findings.

CASE REPORT

A 72-year-old female was found unconscious at 4 P.M. on January 12, 1976. She had last been seen awake at 12 P.M. On admission she was noted to have blood pressure 30/0 and pulse of 78. Caloric and Doll's eye reflexes were negative. She did not respond to pain and had no spontaneous movements. The patient required pressor therapy and mechanical ventilation.

Blood samples tested by gas-liquid chromatography are recorded in Table 1. Purified metabolite was not available for quantitation. The levels listed are based on the glutethamide standard, and should be considered in relative terms rather than absolute amounts.

The patient was in coma for 48 hours and gradually recovered between 48 to 60 hours after which she was discharged.

TABLE 1

Blood Levels of Glutethamide and Its Metabolite

Date	Time	Glutethamide (mg/ml)	Metabolite
1/12/76	5:45 p.m.	35	8
	11:00 p.m.	12	13
1/13/76	8:00 a.m.	10	20
	8:00 p.m.	9	15
1/14/76	11:00 a.m.	1.1	15
	11:00 p.m.	4	10
1/15/76	8:00 a.m.	2	6

Comment

From the table it can be seen that the patient remained in coma long after she had subtoxic levels of glutethamide (toxic range 10-80 mg./ml). Recovery occurred during the descending portion of the curve defined by the relative levels of 4-Hg.

The effect of glutethamide cannot be entirely excluded but the data indicate that the level of 4Hg should be followed in evaluating coma.

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Arthritic symptoms with carcinoid of the ovary: a case report

Franklyn M. Newmark, MD, and Hugh J. McGee, MD, Wheatridge, Colorado

In 1974, Qizilbash et al. reported the forty-seventh case of carcinoid of the ovary in the English literature and reviewed previously reported cases. Of these, twenty-four had the carcinoid syndrome. We wish to report a functioning carcinoid tumor of the ovary, in which arthritic symptoms were prominent.

CASE REPORT

A 54-year-old white woman was first seen in April 1975 complaining of "allergic reactions to food and drinks" since 1970. Her symptoms consisted of flushing of the skin, nasal congestion, swelling of throat and anus, abdominal cramps and diarrhea, cold sweats and articular pain and swelling. The flushing had an immediate onset and lasted minutes to hours. The joint symptoms had a slow onset and would last up to eighteen hours. The flushing was provoked by any kind of alcohol, frankfurters, sausage, pizza, Worcestershire sauce and oranges. The joint symptoms were provoked by oranges, strawberries and peaches. The history in regard to provocative foods was deemed reliable. During the five years of this condition the symptoms were becoming more frequent and more severe. The articular symptoms, consisting of pain, redness and swelling of various joints (wrist, knee, shoulder) had started four months prior to her initial visit. The spectrum of provocative foods seemed to be widening.

Past history included appendectomy 1942, cholecystectomy 1966, repair of umbilical hernia, mild seasonal hayfever, perennial post-nasal drip and anosmia, hives occurring frequently in childhood, one episode of angioedema 1965, one thyroid extract for alleged underactive thyroid, taking allopurinol for alleged gout, and an enlarged uterus noted January 1975.

Family history showed her mother had arthritis (type not known) and her father, brother and sister had asthma and hayfever.

Physical examination showed a mature, overweight, white female with no abnormalities except a pelvic mass thought to be an enlarged uterus. The was no wheezing and no heart murmur. Sigmoidoscopic examination showed no changes except for the pelvic mass extrinsic to the colon. A provocative ingestion test, in the office, with alcohol, produced severe flushing.

Blood count, sedimentation rate and SMA-12 were normal. The serum uric acid determination repeated after withdrawal of allopurinol was 5.0 mgm %. Urine 5HIAA screening test was positive. Blood serotonin (normal 50-200 ng/ml) on 5-3-75 was 97 ng/ml and on 5-29-75 was 694 ng/ml. Radiographic examinations including upper gastrointestinal, small bowel, colon and hypotonic duodenography showed no pathologic changes except for a large hiatus hernia and displacement of the sigmoid colon. Liver scan was normal.

Surgery performed on July 7, 1975 demonstrated no abnormality in the abdomen or pelvis except for an 8 x 10 cm solid tumor of the left ovary, a small cyst in the right ovary and a nodular normal size uterus. Hysterectomy and bilateral Salpingo-oophorectomy were performed. Postoperatively the urine was negative for serotonin on two occasions.

The left ovary consisted of a solid tumor measuring 105 x 85 x 75mm and weighing 200 grams. Cut surfaces were firm, homogeneous and pale pink-tan. Histologically, the tumor was composed of uniform cells arranged in trabeculae, sheets, solid clusters, tubular structures, and rosettes. Nuclei were round with coarse chromatin. Cytoplasm was poorly defined and granular. Fontana Masson stain for argentaffin granules was positive. Electron microscopy showed characteristic electron dense secretory granules in the cytoplasm. No teratomatous element was demonstrated. The uterus contained whorls and cords of smooth muscle fibers. The cyst of the right ovary was lined with granulosa cells. A small nodule attached to the right ovary contained whorls and cords of fibroplastic cells.

The postoperative course was marked by no further episodes of flushing. However, the patient experienced severe arthritis for two weeks that responded to phenylbutazone, with subsequent gradual spontaneous remission over a three month interval. Uric acid was 5.0 mgm % and rheumatoid factor was negative in August 1975. On October 8, 1975 she had an episode of angioedema of the face and throat and generalized urticaria. Allergy to Macademia nuts was suspected. She was successfully

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treated with epinephrine and diphenhydramine. On October 15, 1975 she ate a Macademia nut with no adverse effect. As of July 15, 1976 the patient is symptom free and takes no medication.

Discussion

On the basis of the positive argentaffin reaction and the presence of polymorphic secretory granules, the tumor may be classified as a carcinoid of midgut derivation. This conclusion is further supported by the presence of increased 5HIAA in the urine and elevation of the blood level of serotonin. Production of the carcinoid syndrome by ovarian carcinoid tumors in the absence of metastases has been explained as caused by discharge of 5-HT from these tumors directly into the general circulation bypassing the liver. The prompt alleviation of symptoms by surgical intervention in this patient supports this hypothesis.

Arthritis has previously been reported associated with the carcinoid syndrome, possibly reflecting a more generalized manifestation of the connective tissue disorder commonly affecting the heart valves and the endocardium.[2,3] Qizilbash et al.[1] reported arterial intimal myoxid fibroplasia in mesoappendiceal arteries of a patient with ovarian carcinoid, carcinoid syndrome, systolic heart murmur, and clinical evidence of right heart failure. No such changes were demonstrated in this case. The clinical improvement in the arthritic symptoms following removal of the tumor does however suggest a relationship to carcinoid syndrome.

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Intramural gas after jejunoileal bypass*

Stanley B. Reich, MD, James Monsour, MD, and Emanuel Salzman, MD, Denver

The acute form of intramural gas is being observed with increasing frequency. It has been associated with inferior or superior mesenteric artery occlusion, volvulus, carcinoma, ulcerative and granulomatous colitis, necrotizing enterocolitis, and leukemia. It frequently indicates the occurrence of an acute life-threatening abnormality of the bowel. Chronic pneumatosis has been recorded in every segment of the gastrointestinal tract. It usually occurs in the small bowel where it is often associated with an underlying systemic disease such as scleroderma or chronic obstructive pulmonary disease.

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We have encountered three instances of acute onset of bowel pneumatosis in patients with small intestinal by-pass surgery. In none of these patients was necrotic bowel or other catastrophe encountered. These cases are reported to indicate that a benign form of pneumatosis can occur in this situation.

CASE REPORTS

Case 1: A 48-year-old woman had an ileocecostomy performed April 1973. She developed intractable vomiting, nausea, epigastric pain. Three weeks after surgery, an X-ray of the abdomen showed pneumatosis of the small bowel with no obstruction (Fig. 1). The symptoms were so severe that she was given surgical exploration. There were no significant findings except pneumatosis.

Symptoms persisted over next few months, as did the pneumatosis. At one point she exhibited free intraperitoneal air (Fig. 2). She was explored again and the by-

Fig. 1. Case 1: X-ray of abdomen showing pneumatosis of small bowel.

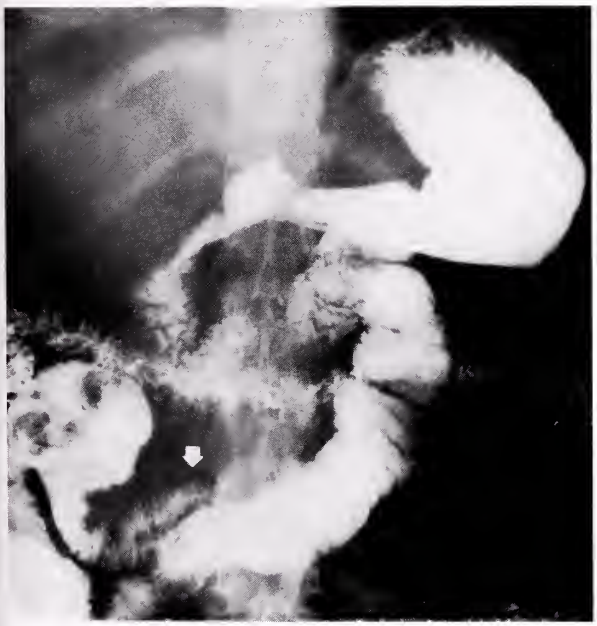

Fig. 3. Case 2: Small bowel series showing pneumatosis of small bowel.

passed small bowel was resected. Most symptoms were relieved. A followup small bowel series several months later showed no pneumatosis. The patient had no evidence of emphysema and had no other findings aside from severe obesity.

Case 2: A 48-year-old obese woman had a small bowel series done eleven months after bypass operation for obesity. This showed a small amount of pneumatosis of the small bowel (Fig. 3). Examination of the small

Fig. 2. Case 1: X-ray of chest showing free intraperitoneal air beneath diaphragms.

Fig. 4. Case 3: Supine film of abdomen showing pneumatosis of cecum and ascending colon.

bowel a week after the surgery had showed nothing. Her mild symptoms of nausea and epigastric pain spontaneously disappeared.

Case 3: A 33-year-old obese woman had jejunoileal bypass in August 1974. She lost 135 pounds. Aside from mild diarrhea, she was asymptomatic until October 1974 when she developed jaundice. At that time a supine film of the abdomen showed pneumatosis of the cecum and ascending colon (Fig. 4). The hepatitis resolved in three weeks. The pneumatosis subsequently disappeared.

Discussion

The evaluation of pneumatosis in the postoperative phase of bypass surgery is difficult. We have encountered three patients with apparently

benign pneumatosis. In a prospective study of ten other patients with bypass surgery, none were symptomatic and none showed pneumatosis by x-ray.

Three theories have been proposed to explain this entity: the mechanical, the bacterial and the biochemical. The mechanical theory states that intestinal gas may dissect into the bowel wall from the lumen, or pulmonary air from ruptured alveoli may dissect through the mediastinum into the retroperitoneal area, along the mesenteric vessels into the bowel wall. The bacterial theory proposes gas producing bacteria within the bowel wall as the source of the cystoides.[12] The biochemical theory suggests excess production of gas in the lumen which is absorbed to form air-filled cysts in the bowel wall; perhaps excessive fermentation of carbohydrates in the small bowel due to disaccharidase deficiency causes severe distention of the bowel with ischemia and necrosis of mucosa and dissection of gas into the bowel wall. In patients with short bowel syndrome, there may be a relative disaccharidase deficiency, and therefore following bypass surgery this mechanism may play a role in the pathogenesis of intramural gas.

Summary

Pneumatosis of the small bowel in the postoperative period following bypass surgery for obesity may occur as a benign complication. It should not be considered a reason for exploration in the absence of other clinical indications.

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NOTE

For a biography of Dr. Alton Ochsner of the Ochsner Clinic, New Orleans, opinions, evaluations, anecdotes, reminiscences, and photos are needed. Photos will be carefully handled and returned. All material gratefully received by Ira Harkey, Ph.D., 401 Metairie Road, #706, Metairie, Louisiana 70005.

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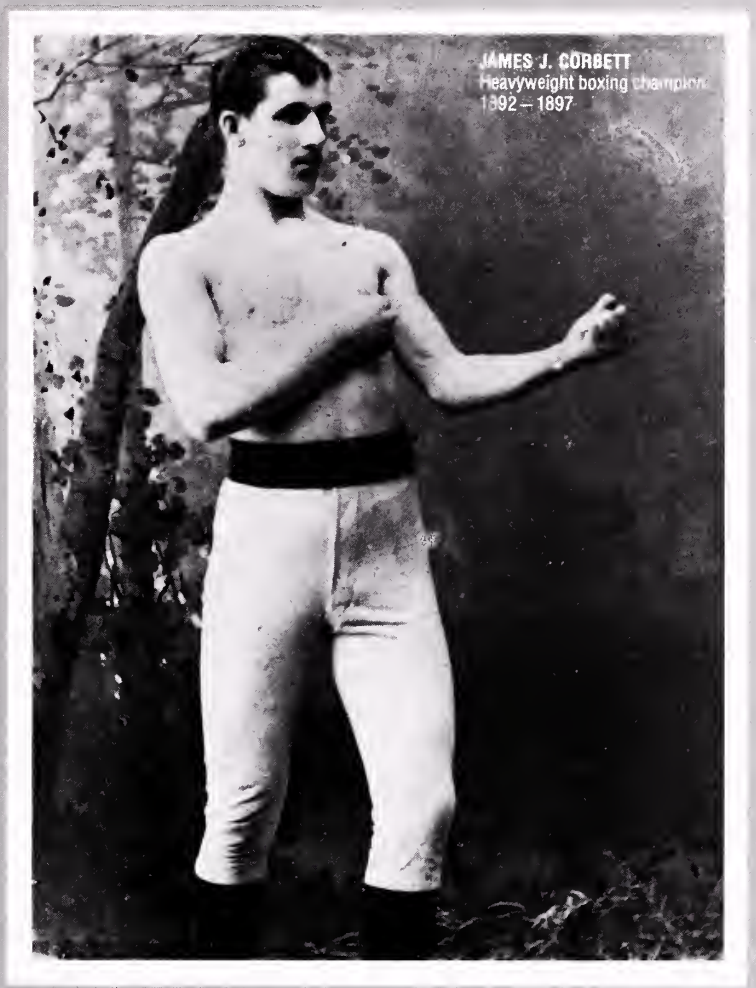

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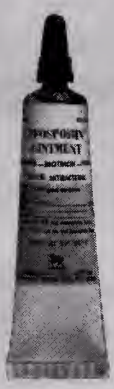

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Wyoming State Medical Society

Seventy-Third Annual Meeting

August 31 - September 3, 1976

Jackson Lake Lodge
Grand Teton National Park, Wyoming

FIRST SESSION

Wednesday, September 1, 1976

The 73rd Annual Meeting of the House of Delegates of the 1976 Wyoming State Medical Society was called to order at Jackson Lake Lodge, Grand Teton National Park, Wyoming, on Wednesday, September 1, 1976, by Dr. Patrick D. Nolan, President of the Wyoming State Medical Society. The President then delivered hsi President's address to the House Delegates.

The President introduced the incoming President of the Wyoming State Medical Society, Dr. James Stoetzel, and turned the meeting over to the Speaker of the House, Dr. Theodore L. Johnston, from Cheyenne.

Dr. Johnston then called for a vote of thanks to the outgoing President, introduced the Assistant Speaker of the House, Dr. Ross Collie, and asked Dr. James Alexander to give a report of the Credentials Committee. A motion was made for acceptance of the credentials report. Motion seconded and carried.

Dr. Johnston then introduced the AMA President, Dr. Richard E. Palmer. Dr. Palmer addressed the House of Delegates. Upon conclusion of Dr. Palmer's speech, Dr. Johnston introduced Mrs. Norman H. Gardner, the President of the American Medical Association Auxiliary, who also addressed the house of Delegates. Dr. Johnston then introduced Mrs. Sally Lund, President of the Wyoming State Medical Society Women's Auxiliary, and Miss Laura Lockhart, President of the American Association of Medical Assistants, both of whom briefly spoke to the House.

Dr. Johnston entered a motion to dispense with the reading of the minutes of the 1975 Annual Meeting. Motion seconded and carried.

Dr. Lee Brubaker then proposed a resolution to ban smoking in the Delegates' meeting. The resolution was referred to the Medical Services Committee. A request was then made for an investigation by the Medical Society into the autopsy laws in the state and potential recommendations for changes. This request was referred to the Executive Government Affairs and Economics Committee.

Dr. Johnston announced there would be a panel of medical education beginning at 11:10 and recessed the first session of the 1976 House of Delegates.

SECOND SESSION

Friday, September 3, 1976

The Second Session of the 73rd Annual Meeting of the Wyoming State Medical Society, House of Delegates, was called to order by Dr. Johnston, Speaker of the House, on Friday, September 3, 1976, at Jackson Lake Lodge, Grand Teton National Park, Wyoming.

Dr. Johnston asked Dr. Kent Christensen for a report from the Time and Place Committee. The committee recommended that the 1977 Annual Meeting be held at Jackson Lake Lodge, August 30 - September 2. A motion was made for acceptance of the Time and Place Committee report. Motion seconded and carried.

Dr. Donald Rohrssen was introduced and presented the report from the Medical Services Reference Committee.

The Annual Meeting minutes of 1975 were read and approved.

The Council meeting minutes of November 9, 1975, were read and approved. Specific attention was directed to Item C with no action required.

The Council Meeting minutes of January 30, 1976, were read and approved.

The Council Meeting minutes of May 2, 1976, were read and approved, specifically Items 5d, 3, 7a were approved.

The 1975 Medical Services Reference Committee Report was read and approved.

The 1975 Executive Government Affairs and Economics Committee minutes were read and approved.

Dr. Rohrssen recommended approval of the above portion of the report. Motion seconded and carried.

Dr. Rhorssen continued with the August 27, 1975, Research Organization Scientific Program Reference Committee report which was read and approved with the recommendation that in lieu of the orientation program a handout should be prepared to be given to prospective members at the time of application for county and state membership. A motion was made for approval of this recommendation. Motion seconded and carried.

Dr. Rohrssen continued with the review and approval of the reports of the Mental Health Committee. A suggestion was made that the next Medical Wire include a notification for a strong review by anyone prescribing Ritalin.

The communication from the Southeast Wyoming Mental Health Center was read and approved with the notation of the favorable impression regarding the continual improvement in the utilization review processes at the state hospital.

The minutes, procedures and budgets of the Rocky Mountain Medical Journal Editorial Board Meeting were read and approved.

Dr. Rohrssen recommended approval of the above portion of the report. Motion seconded and carried.

The letter of Dr. Paul J. Preston, Chairman of the Wyoming Workmen's Compensation Advisory Committee, was read and approved with a commendation for the Committee's excellent service to the practicing physicians of the state.

The special committee report of the Wyoming State Medical Society - Blue Shield Fee Schedule Committee was read and approved with a commendation for Dr. Mahnke and his committee for their outstanding efforts on behalf of the practicing physicians of the state.

Dr. Rohrssen noted that four individuals requested an audience before the Reference Committee: Dr. Marjorie Langman gave a report on the August 9, 1976, meeting of

the Joint Practice Commission and Miss Martin of the State Cervical Cancer Detection Program presented a protocol for her program.

Dr. Rohrssen recommended approval of the above portion of the report. Motion seconded and carried.

The next person to appear before the committee was Miss Laura Lockhart, President of the American Medical Association of Medical Assistants who was seeking approval of the Wyoming State Medical Society through their House of Delegates for their association. After discussion, the committee recommended appointment of a liaison committee to work with the Association of Medical Assistants and report back after investigation.

Motion was made that this portion of the report be approved, motion seconded and carried. Vote taken on approval to instruct the president to appoint a liaison committee, vote carried.

The last person appearing before the committee was Dr. Hobson, whose concern was the method of obtaining an autopsy. After discussion, the committee recommended to the House of Delegates that a task group be formed to propose model legislation and explore the feasibility of having all death certificates signed by a physician.

Motion was made and seconded for acceptance of this portion of the report. After discussion, motion was carried.

Dr. Rohrssen then reported that the resolution to the House of Delegates from Dr. Louis R. Hoyer was read and approved:

> "WHEREAS optometry is attempting to legislate itself into the practice of medicine, by sponsoring and promoting an amendment to Wyoming Statute 33-202, 33-200(a) and 33-304.30; which if passed would enable optometrists to use topical parasympathomimetic, anto cholinergic and sympathomimetic agents. Be it resolved that Wyoming State Medical Society opposes actively and formally such legislation."

The Committee recommended an addition to this that legislative committees throughout the state actively work in opposing this legislation and asked the House of Delegates for a do pass with this addition. Motion made, seconded and carried.

The use of bioptic telescopic lenses while driving was discussed.

> "WHEREAS the visual limitations created by wearing bioptic telescopic lenses while driving has been documented, be it therefore resolved that applicants with these prosthetic aids should not be licensed to drive a motor vehicle except upon individual review and evaluation by the Medical Advisory Board to the licensing agency and after the applicant has demonstrated functional capbaility through a road test."

The committee recommended a do pass from the House of Delegates.

Dr. Rohrssen asked for approval of this portion of the minutes. Motion seconded and carried.

A discussion was then held on the resolution which read "The encroachment by untrained health care practitioners into the practice of medicine". The committee recommended that a standing committee to include members whose fields are most subject to encroachment be appointed to consider rewording of the resolution and opposition of any form of suboptimal patient care by non-physician personnel.

Dr. Rohrssen asked for approval of this portion of the minutes. Motion seconded, discussion held and motion carried.

Dr. Rohrssen then reported that the resolution that no smoking be permitted at the House of Delegates meeting be referred to the House of Delegates with no recommendation. The resolution was handled as a motion from the floor that no smoking be permitted either this year or in years to come until altered by the will of the majority. Motion made, seconded, discussed. Motion to table made, voted upon, not carried. Original motion carried.

Dr. Rohrssen asked for approval of the complete report of the **Medical Services Reference Committee** as amended. Motion was made for approval as amended. Motion seconded and carried.

Dr. Johnston then asked Dr. John Budge, Chairman of the Executive, Government Affairs and Economics Reference Committee, for the panel's report:

The minutes of the Annual Meeting of 1975 were approved. The Council Meeting minutes of August 3, 1975, were reviewed and approved as follows: Items 5b, c, d; 7a(2); 8c(1) and (2); 9d; 11a, b, c, d and f were read and approved. The Council Meeting minutes for November 9, 1975, were reviewed and approved as follows: Items 5a and b; 7c; 8c; 9d; 11a, b, c, d; 12a, b and c were read and approved. The Council Meeting minutes dated August 1, 1976: Items 5c, d; 10b, c, e, f; 11a were read and approved. **Medical Services Refcrence Committee** report dated August 27, 1975; **Resarch Organization and Scientific Program Reference Committee** dated August 27, 1975; and the Executive and Government Affairs Committee report dated August 27, 1975, were approved.

Dr. Budge asked for acceptance of this section of the report. Motion was made, seconded and carried accepting this portion of the report.

Under standing committee reports, information on going without professional liability insurance was presented and was given to the membership by mail. There was a review of legislation, including Enrolled Act No. 2 of the Senate, Enrolled Act No. 8, Enrolled Act No. 15 from the Senate, Enrolled Act No. 13 of the Senate and the Medical Malpractice Reporting Law from the Senate with the exception of the Informed Consent Bill, all of which are presently in effect as law. The report was accepted and motion made for approval, seconded and passed.

Under **Task Force Committee Reports**, William F. Flick, M.D., gave a report from the **Medical Liaison Committee** with special report from the Medical Legal Relationship in Laramie County. A motion for acceptance was made, seconded and passed.

Dr. Budge asked for acceptance of this section of the report. Motion was made, seconded and carried.

A report of the election of the Wyoming Health Services Company, Inc., was presented. Motion was made, seconded and passed that the report received. Dr. Budge asked for acceptance of this section of the report. Motion made, seconded and carried.

A resolution relative to a proposed amendment to the Constitution of the State of Wyoming was read in total. The committee recommended that the resolution become a do pass with the following exceptions: "Areas where mention of $10,000 appears be removed and that no figure relative to the amount of damages be made, assuming that there is no legal or practical reason for inclusion of the figure." Dr. Budge moved that this be accepted by the House of Delegates. Motion seconded, discussion held and motion carried.

The usual, customary and reasonable methodology proposal was read, discussed and accepted. Dr. Budge asked that this portion of the report be accepted. Motion seconded, discussed and carried.

Resolution for a safer Wyoming as proposed by the Wyoming Safety Counell was presented and recommendation for a do pass made. Dr. Budge moved for acceptance of this resolution. Motion seconded and carried.

The renewal questionnaire from Aetna Medical Professional Liability Policy was diseussed and no specific recommendations were made. The State of Wyoming Senate File with regard to provision of medical malpractice insurance through a compensation fund was discussed. The idea was accepted and approved for a do pass.

Dr. Budge asked for acceptance of this portion of the report. Motion seconded and carried.

The committee discussed the publication called **The Drinking American** and recommended that the Wyoming State Medical Society give $1,000 toward production of this in the Casper paper under the alcohol abuse program with the provision to withdraw if the co-sponsors are not felt to be compatible.

Dr. Budge asked for acceptance of this portion of the report. Motion seconded, discussion held and motion carried.

The committee considered the resolution with regard to the optometrists attempt to legislate themselves into the practice of medicine and recommended that the Wyoming State Medical Society oppose actively and formally such legislation.

Dr. Budge asked for acceptance of this portion of the report. Motion seconded, discussion held and motion carried.

Resolution for the Wyoming Legislature from the Wyoming State Medical Society for commendation was read and approved.

Dr. Budge asked for acceptance of this portion of the report. Motion seconded and was carried.

A discussion was held, as a point of interest, with regard to the question of coroners and autopsies.

Motion was made for acceptance of the report in its entirety as corrected. Motion seconded and carried.

Dr. Johnston then called upon Dr. James Alexander for the report of the **Research, Organization and Scientific Program Reference Committee.**

Minutes of the Council Meeting of August 3, 1975, were read and section pertinent to the committee approved as published. Council Meeting minutes of August 29, 1975, were read and sections pertinent to the committee approved as published. The Council Meeting minutes of November 9, 1975; January 30, 1976; May 2, 1976; and August 31, 1976, were read, reviewed and approved as published by the committee. The 1976 secretary's report was read and approved. The executive secretary's report from September 1, 1975, to July 15, 1976, was read and recommended for approval by the committee. Wolf & Company financial report verifying an audit of the Wyoming State Medical Society was read and reeommended for approval. The Wyoming State Medical Society report of income and expenses ending December 31, 1975, and the statement concerning the professional liability assessment was read and recommended for approval. The WYOPAC financial statement was read and recommended for approval. The Wyoming State Medical Society's treasurer's report, financial statement and review of their vouchers was read and recommended for approval.

Dr. Alexander asked that this portion of the report be accepted. Motion seconded and carried.

The committee then considered amendments to the Constitution of the Wyoming State Medical Society as follows:

> Article IX—Fund Dues and Expenses. Section 1, Line 4—Delete the word "March" and substitute the word "January".
>
> Article IV—Membership, Section 2, concerning the deleting.
>
> Article IV, Section 2(f)—Deleting the orientation program and adding the qualifications for membership to the Wyoming State Medical Society. To pass the flex test and to be entered in this or shall have a degree from an accredited school of osteopathy.

Dr. Alexander asked for acccptance of this portion of the report. Motion seconded and carried.

The **Long Range Planning Committee** minutes of August 31, 1976, were reviewed and recommended for approval. A motion was carried that the program on medical education presented following the opening delegates meeting be accepted as the report of the **Medical Education Committee.** The minutes of the Reference Committee reports of August 27, 1975, were read and recommended for approval. The Memorial Subcommittee report was read and recommended for approval.

Dr. Alexander asked for acceptance of this portion of the report. Motion seconded, discussion held and motion carried.

The president's address to the House of Delegates was read and the committee recommended commendation to Dr. Patrick Nolan for his good leadership during the year 1976.

Dr. Johnston then called for the report of the Nominating Committee and the Election of Officers. The following nominations were made: for President, Dr. James Stoetzel; for President-Elect, Dr. Kent Christensen; for Vice President, Dr. James Alexander; for Secretary, Dr. John Budge. A unanimous vote was cast for these officers. Nominations for Treasurer were Drs. Donald Rhorssen and Arnie Krause, and Dr. Rohrssen being elected. Dr. Johnston called for nomination of the Speaker of the House of Delegates, receiving the name of Dr. Ross Collie. After calling for additional nominations and receiving none, Dr. Collie was elected. Dr. Richard McCleery was nominated for Vice Speaker. After calling for and receiving no additional nominations, Dr. McCleery was declared Vice Speaker. Dr. Fenworth Downing was nominated for delegate to the AMA and Dr. John Corbett was nominated as alternate delegate. After calling for additional nominations and receiving none, Dr. Johnston declared the candidates elected by acclamation.

A motion was made, seconded and carried that the president be requested to appoint six men as members of the **Long Range Planning Committee.** The appointments were: from the Northwest Society, Dr. Lee Herman; Sheridan, Dr. Bob Henderson; Teton, Dr. Bill Fogarty; Campbell, Dr. Paul Kauffman; Rock Springs, Dr. Rudy Lansang; Fremont, Dr. John Russo.

Dr. Patrick Nolan recommended that the House of Delegates give Dr. Johnston a commendation on his excellent work as Speaker of the House during the past four years.

Dr. Johnston adjourned the 1976 Meeting of the State Medical Society House of Delegates.

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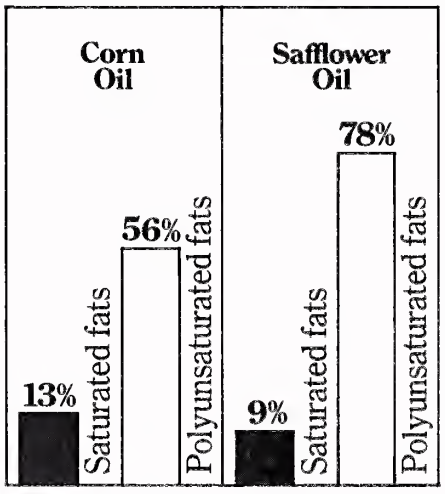

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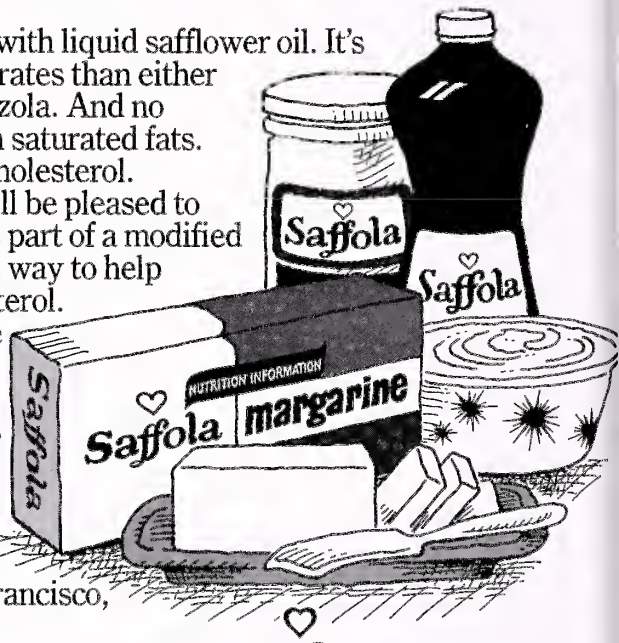

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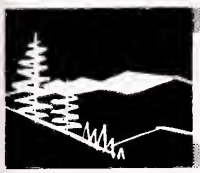

COLORADO

Study of Medical Therapy of Crohn's Disease

Sulfasalazine and prednisone have been found to be the most useful and effective drugs for treatment of patients with active symptomatic Crohn's disease (regional enteritis). This conclusion from Part I of the National Cooperative Crohn's Disease Study sets the background for a current study which will enroll patients through March, 1977. This study, entitled a Trial of Adjunctive Therapy in Crohn's Disease, will address the question "Is the combination of the two drugs together better than prednisone alone for the treatment of Crohn's disease?"

In eight study centers across the country (including one at the University of Colorado Medical Center) there is a continuing need for patients actively suffering from Crohn's disease to participate in the current study. They will receive either prednisone alone, or combination prednisone and sulfasalazine. Patients who improve in the first eight weeks of either treatment will continue to be followed for six months during which an attempt will be made to withdraw prednisone.

The study is "double-blind, randomized and prospective." In a double-blind study, neither the patient or his physician is aware of which drug regimen he is receiving. A randomized study is one in which the assignment of patients to one or other treatment regimen is made by random choice. And "prospective" means that observations are made in an ongoing, day-to-day fashion according to a predetermined protocol. To assure that drug dosage is tailored to the patient's needs and to guard against possible drug side effects, while at the same time maintaining the "double-blind" of both patient and physician, a second physician prescribes the study medications. He does not encounter the patient, but prescribes dose based on information gathered at each visit from the patient, the laboratory, and the patient's physician.

Another unique feature of the study is use of the Crohn's Disease Activity Index, an eight-item numerical score measuring both subjective and objective factors. The index value is calculated at each patient visit, and based on it the response to treatment is judged and the dose of prednisone adjusted.

Rocky Mountain physicians are asked to contact the Denver Study Center about patients with Crohn's disease whom they think might benefit from evaluation for and participation in the study. Call or write to John W. Singleton, M.D., 4200 E. 9th Avenue, Denver, CO 80220; (303) 394-7697.

Smyth Honored

Charley J. Smyth, MD, professor of medicine at the University of Colorado Medical Center and head of the Joe and Betty Alpert Arthritis Treatment Center at General Rose Memorial Hospital, received the Hospital's Award of Honor for "distinguished service to medicine and humanity."

Colorado Doctors Named to ACP

Richard W. Moothart, MD of Colorado Springs, James H. Ellis, Jr., MD, and Steven A. Sahn, MD of Denver were elected Fellows of the American College of Physicians at a recent meeting of the ACP Board of Regents in Philadelphia. These doctors have won their Fellowships through scientific accomplishments and by recognition of their leadership in their specialties as determined by fellow practitioners.

Yoder Named to Serve

Franklin Yoder, MD, Greeley doctor, has been reappointed to the Residency Review Committee for Preventive Medicine of the American Medical Association, and has been elected to serve as Vice Chairman of the Committee for the coming year.

Jackson, Mimmack Receive CU Honors

Ham Jackson, MD, Fort Morgan, Colorado, and Frederick Mimmack, MD, Denver received distinguished clinical faculty awards at the annual medical staff dinner of the University of Colorado hospitals in December. Dr. Jackson received his MD in 1951 from the CU School of Medicine, and since 1964 has been a clinical faculty member; currently he is an assistant clinical professor of medicine. Dr. Mimmack became an assistant is psychiatry at UCMC in 1960 and is now an assistant clinical professor of psychiatry.

Cardiologists Honored

Laurence DeK. Favrot, MD, and Robert F. Grover, MD, both of Denver, have been granted Fellowship in the American College of Cardiology, iulfilling stringent membership requirements, which reflects the esteem of their colleagues.

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* **Indications:** When the fixed combination represents the dosage determined by titration: Adjunctive therapy in edema associated with congestive heart failure, hepatic cirrhosis, the nephrotic syndrome. Corticosteroid and estrogen-induced edema, idiopathic edema; hypertension, when the potassium-sparing action of its 'Dyrenium' component is warranted.

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Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in severely ill patients with urine volume less than one liter/day, the elderly or diabetics, with suspected or confirmed renal insufficiency. Periodic determinations of serum K^+ should be made. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **The presence of a widened QRS complex or arrhythmia in association with hyperkalemia requires prompt additional therapy.** Thiazides are reported to cross the placental barrier and appear in breast milk; fetal or neonatal hyperbilirubinemia, thrombocytopenia, altered carbohydrate metabolism and other adverse reactions that have occurred in the adult may result. When used in pregnancy or in women who might bear children, weigh potential benefits against possible hazards to fetus. Adequate information on use in children is not available.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics, or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving Dyrenium® (triamterene, SK&F Co.), and leukopenia, thrombocytopenia, agranulocytosi and aplastic anemia have been reported wit thiazides. Do periodic blood studies in cirrhoti to check for nondrug-related variations in bloc pictures, and in patients with folic acid depletio since 'Dyrenium' may contribute to appearan of megaloblastosis. Antihypertensive effect ma be enhanced in post-sympathectomy patient Use cautiously in surgical patients. The followir may occur: transient elevated BUN or creatinir or both, hyperglycemia and glycosuria (diabet insulin requirements may be altered), hyperur cemia and gout, digitalis intoxication (in hyp kalemia), decreasing alkali reserve with possib metabolic acidosis. 'Dyazide' interferes wit fluorescent measurement of quinidine.

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MEETINGS

Colorado Ophthalmological Society

6TH ANNUAL MIDWINTER SEMINAR IN OPHTHALMOLOGY

The Lodge, Vail, Colorado

January 29 - February 2, 1977

Contact: Mrs. Shirlee Meyers, 1601 East 19th Ave., Denver, Colorado 80218

National Asthma Center

NEW DEVELOPMENTS IN ALLERGY AND IMMUNOLOGY

Keystone Ski Resort, Colorado

January 30 - February 5, 1977

Contact: Hyman Chai, MD, National Asthma Center, 1999 Julian St., Denver, Colorado 80204.

University of Colorado School of Medicine Continuing Medical Education

NEWBORN AND PEDIATRIC RADIOLOGY

Given Institute of Pathobiology, Aspen, Colorado.

February 7 - 11, 1977

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3RD ANNUAL WINTER SKIN SEMINAR

Given Institute of Pathobiology, Aspen, Colorado.

February 14 - 18, 1977

* * *

RHEUMATOLOGY

Given Institute of Pathobiology, Aspen, Colorado.

February 21 - 25, 1977

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NATIONAL EMS SYMPOSIUM — CLINICAL TOXICOLOGY

Humphreys Postgraduate Center, Denison Library Building, University of Colorado Medical Center

March 1 - 4, 1977

SEXUAL AMBIGUITY

Given Institute of Pathobiology, Aspen, Colorado.

March 7 - 11, 1977

* * *

FAMILY PRACTICE REVIEW

Humphreys Postgraduate Center, Denison Library Building, University of Colorado Medical Center

March 7 - 12, 1977

* * *

3RD ANNUAL PEDIATRIC REVIEW

Humphreys Postgraduate Center, Denison Library Building, University of Colorado Medical Center

March 19, 1977

* * *

SECOND WINTER HEMATOLOGY - ONCOLOGY MEETING

Given Institute of Pathobiology, Aspen, Colorado

March 21 - 25, 1977

Contact: Office of Postgraduate Medical Education, University of Colorado Medical Center, Container C295, 4200 East 9th Avenue, Denver, Colorado

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University of New Mexico School of Medicine

3RD ANNUAL ROCKY MOUNTAIN NEURO-OPHTHALMOLOGY COURSE

Tamarron, Durango, Colorado

February 9 - 11, 1977

Contact: Thomas J. Carlow, MD, c/o Neuro-Ophthalmology Course, Department of Neurology, University of New Mexico, 2211 Lomas Blvd., NE. Albuquerque, New Mexico 87131

Colorado Trudeau Society

16TH ANNUAL JOHN R. DURRANCE MIDWINTER CHEST CONFERENCE

Aspen Institute, Aspen, Colorado

February 10 - 12, 1977

Contact: Colorado Trudeau Society, 1600 Race St., Denver, Colorado 80206

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This five-day multidisciplinary course is a concise but comprehensive review of basic medical science and is designed to meet the needs of those clinicians who wish to expand their knowledge of molecular biology. Early registration is advised as attendance is limited. Tuition fee is $250 payable upon application for enrollment.

General lectures will cover a review of chemistry and physics for clinicians, chemical bonds of biological importance, subatomic particles, correlation of cell structure and function, the structure and function of proteins, sugars and lipids, the structure of nucleic acids, DNA synthesis, expression of genetic information, regulation of gene expression, evolution of proteins, carbohydrate and fat metabolism, bioenergetics, molecular aspects of visual excitation, the organization and function of the brain, mutagenic hazards.

Elective sessions will include the biology of water, experimental discovery of charm, bacterial antibiotic resistance, salt and water, interferon, genetic manipulation of microorganisms, dysoxia, association between HL-A and various disease states, muscle contraction, nutrition, prostaglandins, atherosclerosis, mechanisms of immunity, extranuclear genetics, genetics: problems and opportunities, nerve growth factor, drug idiosyncrasy, molecular mechanisms of drug action.

A faculty of 21 Stanford University biomedical scientists, including four Nobel Laureates, will take part in the program.

THERAPY: PRINCIPLES AND USE OF DRUGS

AT STANFORD UNIVERSITY • MARCH 28-APRIL 1, 1977

The objective of this five-day course is to familiarize physicians, and others involved in clinical pharmacology, with the concepts of modern drug use. The program includes an in-depth review of the basic principles and the practical aspects of drug action. **The course is intended to assist physicians in their daily clinical work.** Tuition fee is $250 payable upon enrollment, and advance registration is required.

Morning lectures will review important principles of chemistry and biochemistry and will deal with molecular mechanisms of drug actions; routes of administration; elimination, metabolism, and time course of drug action; toxicity and idiosyncrasy; tolerance and physical dependence; teratogenesis, mutagenesis, carcinogenesis; and drug interactions. Afternoon informal seminars will cover the rapidly changing field of applied pharmacology.

Twenty-seven faculty members of the Stanford University School of Medicine will participate.

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Tuition fee is $275, payable with $120 room deposit to Stanford University School of Medicine upon application for enrollment. Acceptance of enrollment assures firm hotel reservation with choice of type of room at prevailing Mauna Kea rates. Group flights departing Saturday, April 30, and returning Saturday, May 7, may be available from San Francisco and Los Angeles, as well as reduced tour airfares from other originating cities. Early application is advised.

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Columbus Hospital

BIG SKY RADIOLOGY CONFERENCE

Huntley Lodge, Big Sky, Montana

February 10 - 12, 1977

Contact: Virgil B. Graves, MD, Department of Radiology, Columbus Hospital, 1601 Second Avenvue North, Great Falls, Montana 59401

University of Utah Medical Center

22ND ANNUAL ANESTHESIOLOGY COURSE

C'est Bon Hotel Convention Center, Park City, Utah

February 11 - 15, 1977

Contact: Department of Anesthesiology, University of Utah Medical Center, 50 North Medical Drive, Salt Lake City, Utah 84132

Albuquerque and Bernalillo County Medical Association

TAOS MEDICAL SYMPOSIUM

Taos, New Mexico

February 16 - 19, 1977

* * *

TAOS SPORTS MEDICINE SYMPOSIUM

Taos, New Mexico

March 6 - 11, 1977

Contact: Thomas A. Bodnar, 2650 Yale Blvd., SE #103, Albuquerque, New Mexico 87106

Beth Israel Hospital

VAIL FAMILY/GENERAL PRACTICE CONFERENCE

The Mark at LionsHead, Vail, Colorado

February 12 - 19, 1977

* * *

VAIL OB/GYN CONFERENCE

The Mark at LionsHead, Vail, Colorado

February 19 - 26, 1977

ASPEN RADIOLOGY CONFERENCE

Aspen Institute, Aspen, Colorado

February 26 - March 5, 1977

* * *

VAIL PSYCHIATRY CONFERENCE

Lion Square at LionsHead, Vail, Colorado

March 5 - 12, 1977

* * *

VAIL GENERAL SURGERY CONFERENCE

The Mark at LionsHead, Vail, Colorado

March 12 - 19, 1977

Contact: Beth Israel Hospital Conference Program, 1818 Gaylord Street, Denver, Colorado 80206

American Thoracic Society, New Mexico Chapter

5TH ANNUAL TAOS LUNG DISEASE SYMPOSIUM

Kachina Lodge, Taos, New Mexico

February 25 - 27, 1977

Contact: Mary Gordon Giblin, 216 Truman Ave., NE, Albuquerque, New Mexico 87108

Stanford University School of Medicine

BASIC SCIENCE FOR CLINICIANS

February 28 - March 4, 1977

Tresidder Memorial Union, Stanford University, Stanford, California

Contact: Office of Postgraduate Medical Education, Stanford University Medical Center, Stanford, California 94305

Loveland - Bataan Medical Center

2ND ANNUAL "SKI AND SUBSTRATE SEMINAR"

March 3 - 5, 1977

Kachina Lodge, Taos, New Mexico

Contact: American Diabetes Association New Mexico Affiliate, 6101 Marble Ave., NE, Albuquerque, New Mexico 87108

AMA Expands Continuing Medical Education Opportunities in 1977.

Now you can choose from 15 regional CME meetings!

Recognizing the importance of continuing medical education to its members, the AMA has greatly expanded its CME programs. During 1977, the AMA will offer 15 regional CME meetings around the country in addition to its scientific programs at both the Annual Convention and Winter Meeting.

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All courses are approved by the AMA Council on Continuing Physician Education for Category 1 credit toward an AMA Physician's Recognition Award. A syllabi written by medical school faculties is provided with every course.

Specific information on course location, fees, academic program, faculty, and hotel reservations will be available approximately 2 months before each course date. Please write to address below at that time stating your selection(s). Print name, address, and office phone number.

1977 Regional Schedule

Tulsa, Oklahoma	January 22-23
Birmingham, Alabama	February 5-6
*Lake Tahoe, Nevada	February 11-13
Denver, Colorado	February 19-20
*Tarpon Springs, Florida	March 4-6
Detroit (Southfield), Michigan	March 26-27
New York (Westchester), New York	April 16-17
Houston, Texas	May 15
Hartford, Connecticut	September 10-11
*Lake of the Ozarks, Missouri	September 16-18
Chicago, Illinois	September 24-25
*Hot Springs (Homestead), Virginia	Sept. 30-Oct. 2
*Huron, Ohio	October 7-9
*Honolulu, Hawaii	Oct. 30-Nov. 4
Hershey, Pennsylvania	November 18-19

AMA's 126th Annual Convention

San Francisco, California	June 18-22

AMA's Winter Scientific Meeting

Miami Beach, Florida	December 10-13

AMA Spokesmanship Seminars

Chicago, Illinois	August 13-14
(Marriott O'Hare Hotel)	November 12-13

*Courses end at midday for recreation activities

AMA Department of Meeting Services
535 North Dearborn Street
Chicago, Illinois 60610

Vail Cosmetic Surgery Society

5TH ANNUAL VAIL COSMETIC SURGERY SYMPOSIUM

March 5 - 12, 1977

The Lodge, Vail, Colorado

Contact: The Lodge at Vail, Vail, Colorado 81657

American Heart Association - San Diego Academy of Family Practice

NON-INVASIVE DIAGNOSTIC TECHNIQUES IN VASCULAR DISEASE

March 14 - 18, 1977

Vacation Village Hotel, Mission Bay Park, San Diego, California

Contact: Vascular Disease Foundation, Box 705, La Jolla, California 92038

American College of Cardiology

"CONTEMPORARY MANAGEMENT OF ACUTE MYOCARDIAL INFARCTION" BY THE FAMILY PHYSICIAN

March 17 - 19, 1977

Hay Adams Hotel, Phoenix, Arizona

* * *

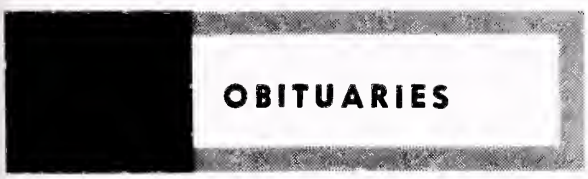
American Academy of Family Physicians, New Mexico Chapter

PURGATORY COLORADO MEDICAL SKI SYMPOSIUM

March 18 - 19, 1977

Ramada Inn, Durango, Colorado

Contact: Albuquerque Academy of Family Physicians, 2650 Yale Blvd., SE, #105, Albuquerque, New Mexico



Colorado

Doctor **Calvin N. Caldwell, MD,** died Thursday, October 14, 1976 in Pueblo, Colorado. Born, December 4, 1891 in Hangchow, China, of missionary parents, and educated at the Shanghai American School, he returned with his family to the United State shortly after the turn of the century to attend Fredricksburg College, Virginia.

He received his MD from the University of Louisville, Louisville, Kentucky, in 1917, and immediately entered the U.S. Navy medical service. At the end of World War I he became health officer in Georgetown, Kentucky, where he remained for about a year before opening an office for general practice in Pueblo, Colorado, where he served both professionally and in civic affairs from 1920 until his retirement in 1972.

Doctor Caldwell played an important role in the founding of Pueblo Junior College and served many years on its Board, serving several years as chairman. He also was coroner of Pueblo County for several terms. He was the local Denver and Rio Grande Railroad surgeon for many years.

He belonged to the Pueblo County Medical Society, serving one year as president, and to the Colorado Medical Society as well as the American Medical Association. He was a Mason, a member of the Pueblo Chamber of Commerce, the Kiwanis Club, the Elks and the Presbyterian Church.

He was married on August 27, 1918 in Louisville, Kentucky to Margaret Mann, and they had two daughters and a son. Surviving are his wife, Margaret, two daughters, Mrs. Annie Venditti, Knoxville, Tennessee, and Mrs. Clara Kvran, Malta, and a son, Calvin N. Jr., Durango, Colorado.

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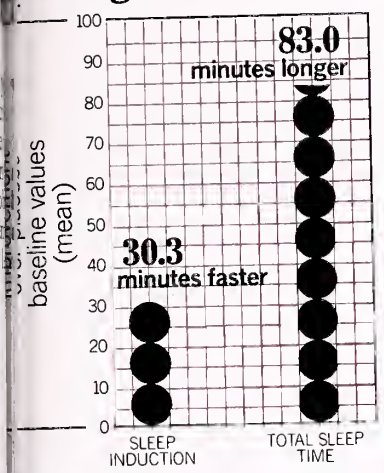
lerly insomniacs asleep faster, ot longer[1]



sults expand and nfirm objective oof of efficacy younger adults th insomnia

he effectiveness of Dalmane razepam HCl) was ionstrated in earlier studies of ounger adults with trouble ng asleep, staying asleep or ping long enough.[2] On average, ese studies, Dalmane induced p within 17 minutes and vided 7 to 8 hours of sleep, at same time reducing number ighttime awakenings.

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Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).
***Usage in Pregnancy:* Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**
Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

REFERENCES:
1. Frost JD Jr: Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
2. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
3. Robinson DS, Amidon EL: Interaction of benzodiazepines with warfarin in man, in *The Benzodiazepines*, edited by Garattini S, Mussini E, Randall LO. New York, Raven Press, 1973, p. 641

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THE BOOK CORNER

New books received are acknowledged in this section and such acknowledgment must be regarded as sufficient return for the courtesy of the sender. Selection will be made for review in the interests of our readers and as space permits. Books are listed with advance data supplied by publishers. Prices quoted are not guaranteed. For further information, address queries to the publishers. Books here listed are available for lending from the Denver Medical Society Library.

A copy of the New Doctor's Kit prepared by the AMA's Practice Management Department is available in the Library. The kit contains the publications Planning Guide for Physicians' Medical Facilities, The Business Side of Medical Practice, Group Practice Guidelines, Preparing a Patient Information Booklet, Talking with Patients, as well as medico-legal forms.

Recent Acquisitions

Arthritis Surgery: Leonard Marmor. Philadelphia, Lea & Febiger, 1976. 548 p.

Diet Manual: Massaehusetts General Hospital, Dietary Department. Boston, Little, Brown & Co., 1976. 156 p.

The Diagnosis of Bleeding Disorders: Charles A. Owen, E. J. Walter Bowie and John H. Thompson, Jr., 2nd ed. Boston, Little, Brown & Co., 1975. 398 p. $21.50.

Differential Diagnosis in Pediatric Otolaryngology: Marshall Strome, ed. Boston, Little, Brown & Co., 1975. 299 p. $19.95.

Early Care of the Injured Patient: American College of Surgeons. 2nd ed. Committee on Trauma. Philadelphia, Saunders, 1976. 443 p. $12.50.

Emergency-Room Care: Charles L. Eckert, ed. 3rd ed. Boston, Little, Brown & Co., 1976. 480 p. $12.50.

Management of the Patient with Cancer: Thomas F. Nealon, ed. 2d ed. Philadelphia, Saunders, 1976. 1012 p. $45.00.

Maturation of the Therapeutic Community: Maxwell S. Jones, New York, Human Scienees Press, 1976. 169 p. $12.95.

Mechanisms of Neurological Disease: Anthony Lewis. Boston, Little, Brown & Co., 1976. 540 p. $20.00.

Practical Ophthalmic Plastic and Reconstructive Surgery: Merrill J. Reeh. Philadelphia, Lea & Febiger, 1976. 205 p.

Principles of Anesthesiology: Vincent J. Collins. 2nd Ed. Philadelphia, Lea Febiger, 1976. 1671 p. $60.00.

Psychiatric Emergencies: Robert Glick, ed. N. Y., Grune & Stratton, 1976. 300 p. $16.00.

Roentgen Diagnosis of Bones: Mogens Eiken. Chicago. Year Book, 1975. 85 p.

The Secret Strength of Depression: Frederic F. Flach. Philadelphia, Lippincott, 1974. 288 p. Gift. $7.95.

Two Centuries of American Medicine: 1776-1976. Philadelphia, Saunders, 1976. 844 p. $19.76.

Wilm's Tumor: Carl Pochedly and Denis Miller, ed. N. Y., John Wiley & Sons, 1976. 239 p. $18.50.

Book Review

Triumphs of Medicine: Edited by Harry Keen, M.D., F.R.C.P., John Jarrett, M.D., and Arthur M. Levy, M.D., London and New Hampshire, U.S.A., 1976.

A fascinating, small folio volume, well illustrated, which takes the reader into the realm of history along with medieval achievements and modern advances in the treatment of various diseases, drawn from many countries.

The volume deals with medicine as practiced by priests through the Golden Age of Greek medicine along with Hippocrates, down to Andreas Vesalius and William Harvey, which led into the field of scientific studies of anatomy, physiology, pathology, and bacteriology. This formed the basis of diagnostic and therapeutic medicine. All of these discoveries led to the conquest of infectious diseases such as smallpox, cholera, and typhoid fever. This was achieved partially through the development and improvement of the microscope.

Knowledge of body chemistry, the heart, high blood pressure, coronary heart disease, vein grafting, cardiac valve replacement, and orthopedic surgery all gave medicine a great impetus as well as rehabilitating the crippled and the sick.

The reader is taken to the brain and spinal cord, with improvements in the diagnosis and treatment of conditions involving the nervous system. Vision and hearing take important places in *Triumphs of Medicine,* along with the organ transplants of kidneys and other viscera. The scope of the volume is broadened to include anesthesia in surgery and drugs used in modern medicine.

This volume belongs in every library, for it ties in history with the great *Triumphs of Medicine* through illustrations throughout the text.

Nolie Mumey, MD
Denver

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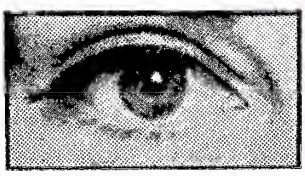

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PEDIATRICIAN needed by 43-man multispecialty group in the Rocky Mountain area. Excellent location. Progressive group. Salary competitive. Full partnership in two years. Write: Paul V. Hoyer, M.D., Medical Director, P.O. Box 2555, Billings, Montana, 59103. 1176-5-4B

MENTAL HEALTH OFFICE to sub-lease in multidisciplinary group in Greater Phoenix. Lease is for 18 months with option to buy. Excellent opportunity for anyone moving to Phoenix. Call: (602) 957-1740. 1176-9-2B

WASHINGTON STATE NEEDS 2 GPs and FPs and 1 General Surgeon. 3 physicians currently on medical staff. Opportunity in rural area, with hunting, fishing, water sports within 5 minutes. Contact: Riehard R. Bell, Executive Director, Coulee Community Hospital, P.O. Box H, Grand Coulee, Washington 99133. Call: (509) 633-1753. 1176-10-3B

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COLORADO PHYSICIANS. Interested in the Physicians Assistant concept? Contact the Colorado Academy of Physieian Assistants, c/o A.V. Ciuffo, PA, 8383 W. Alameda Avenue, Lakewood, Colorado 80226. 1176-28-3B

FAMILY PRACTITIONER IN Windsor desires an associate. Town of 3300. Service area of 5,000. Rapidly growing community 50 miles north of Denver. 14 miles to Greeley. Contact: Edwin D. Kadlub, MD, P.O. Box 250, Windsor, Colorado 80550, or call: (303) 686-7414. 1176-29-TFB

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NOTE

Contacts with physicians who have had personal contacts with those who have suffered from Munchausen (Hospital Addiction) Syndrome or variants are sought by S.E. Hyler, MD, 5620 Netherland Avenue, Riverdale New York 10471.

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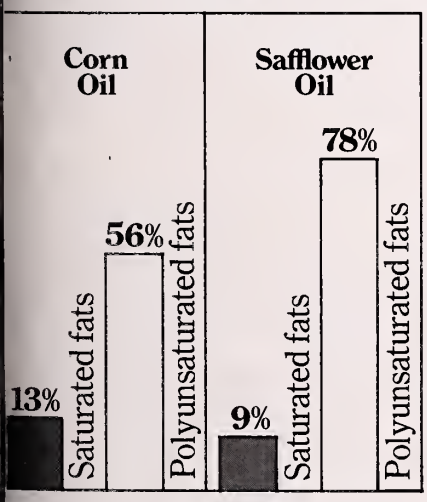

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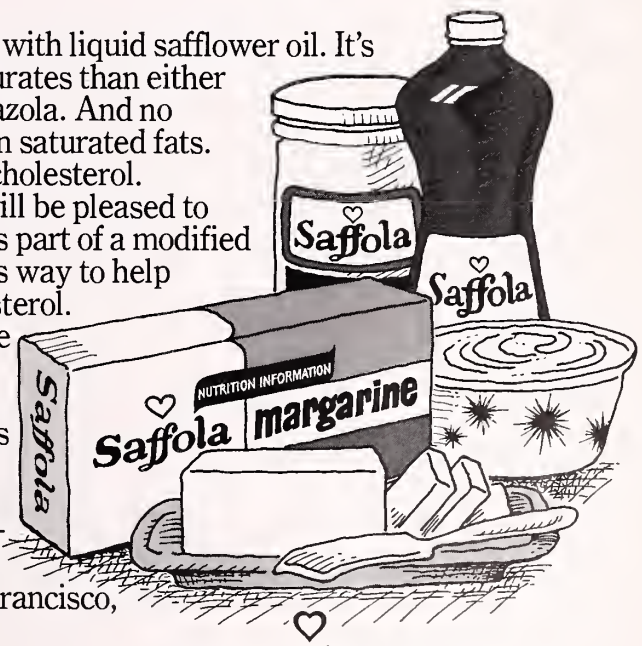

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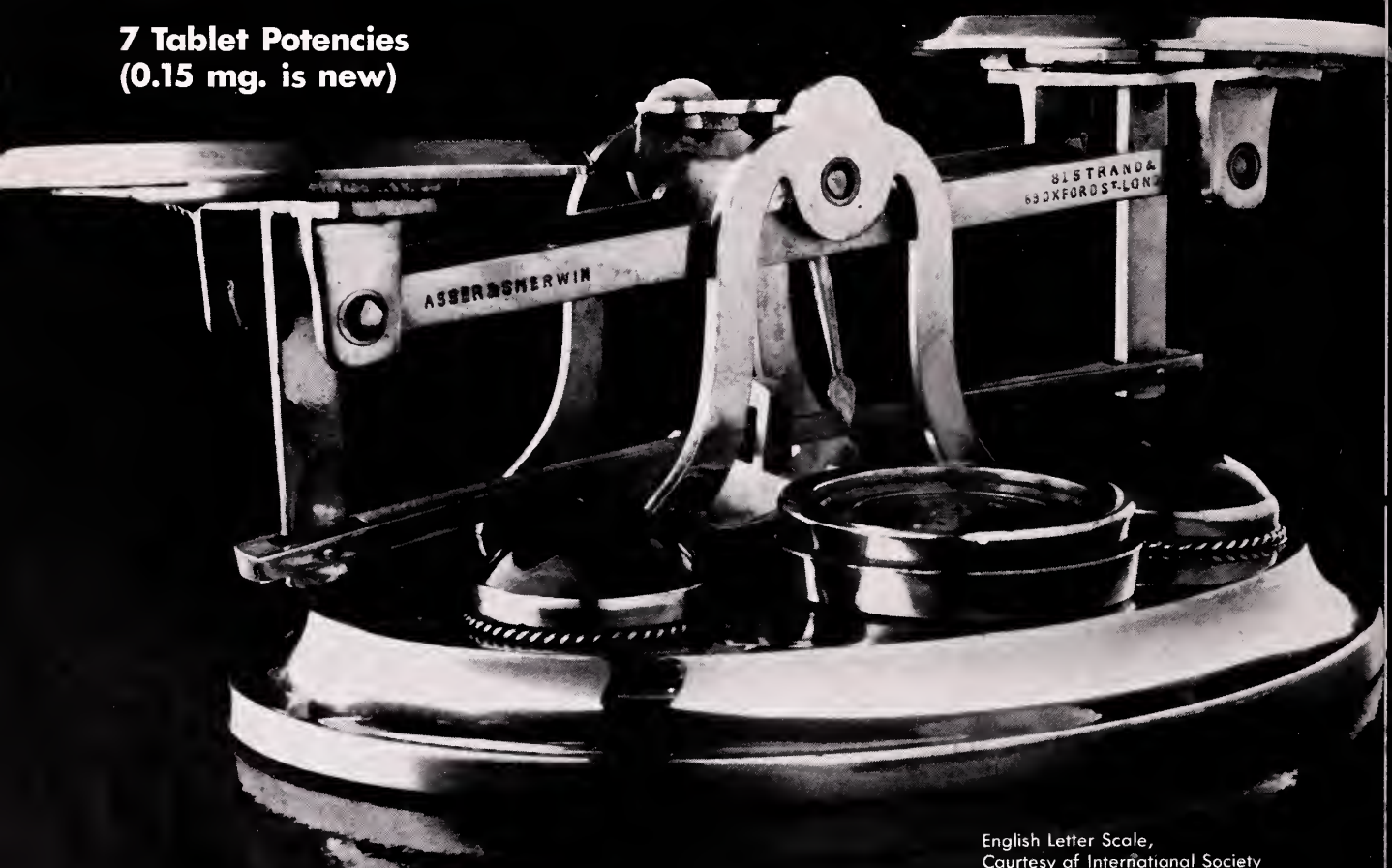

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Warnings. Drowsiness may occur. PHENOBARBITAL MAY BE HABIT-FORMING.

Precautions. Use with caution in the presence of cardiovascular disease, severe hypertension, hyperthyroidism, prostatic hypertrophy, or glaucoma.

Adverse Reactions. Mild epigastric distress, palpitation, tremulousness, insomnia, difficulty of micturition, and CNS stimulation have been reported.

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in Brief

A summary of AMA, medical & health news

American Medical Association / 535 North Dearborn Street
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The AMA's national health insurance bill, titled "The Comprehensive Health Care Insurance Act of 1977," is number HR 1818 in the House and S 218 in the Senate. At a news conference following introduction of the bill in Congress recently, AMA President Richard E. Palmer, MD, said the proposal "fairly represents the viewpoints of this country's private sector" and "is a viable solution to the problems of providing quality medical care to all Americans." Joseph Califano announced at hearings on his confirmation as secretary of HEW that no national health insurance proposal is planned by the Carter Administration this year.

The most violent TV network last fall was the National Broadcasting Company, according to the National Citizens Committee for Broadcasting, which received a $25,000 grant from the AMA to support its monitoring of violence on TV. The American Broadcasting Company was second and the Columbia Broadcasting Company was the least violent.

An administrative law judge has certified to the full Federal Trade Commission the AMA's motion for FTC reconsideration of its restraint of trade complaint. In addition, the FTC administrative law judge has changed the trial date from June 7 to Sept. 7. The FTC complaint, regarding advertising by physicians and medical ethics, was issued against the AMA, the Connecticut State Medical Society and the New Haven Medical Assn. in December, 1975.

The 30th National Conference on Rural Health will be March 30-April 1, at Washington Plaza, Seattle. The theme for the meeting will be "Directions for Rural Health Services."

The FTC was asked to reconsider its complaint of restraint of trade against the AMA and two medical societies regarding advertising by physicians. In a brief filed with the commission, the AMA said its policy book on ethics, *Opinions and Reports of the Judicial Council*, was being revised before the FTC complaint was filed. As a result, the brief said, the AMA is being forced to defend statements that do not even reflect current policy. The FTC complaint was filed in December, 1975, against the Association, the Connecticut State Medical Society and the New Haven Medical Assn.

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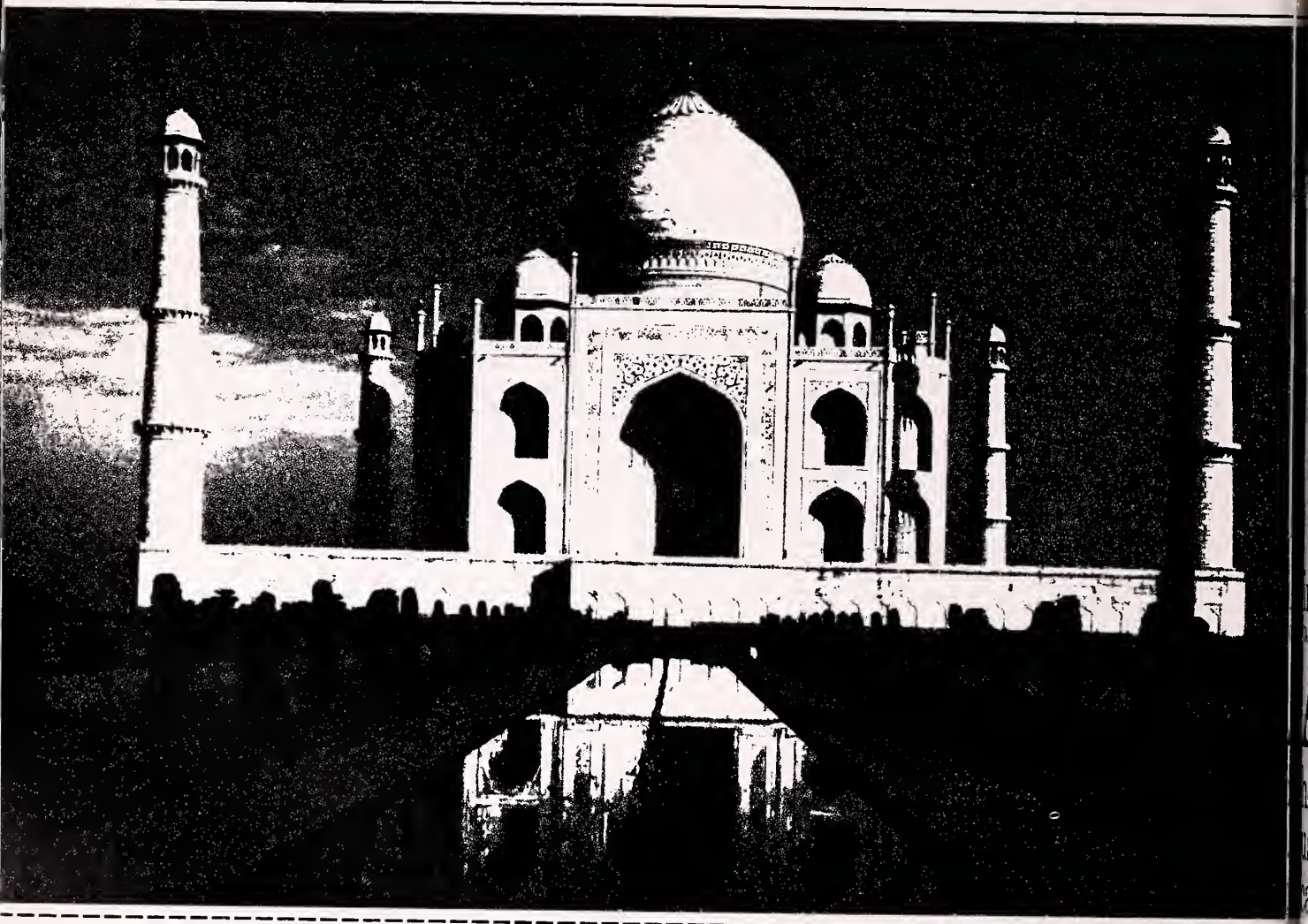

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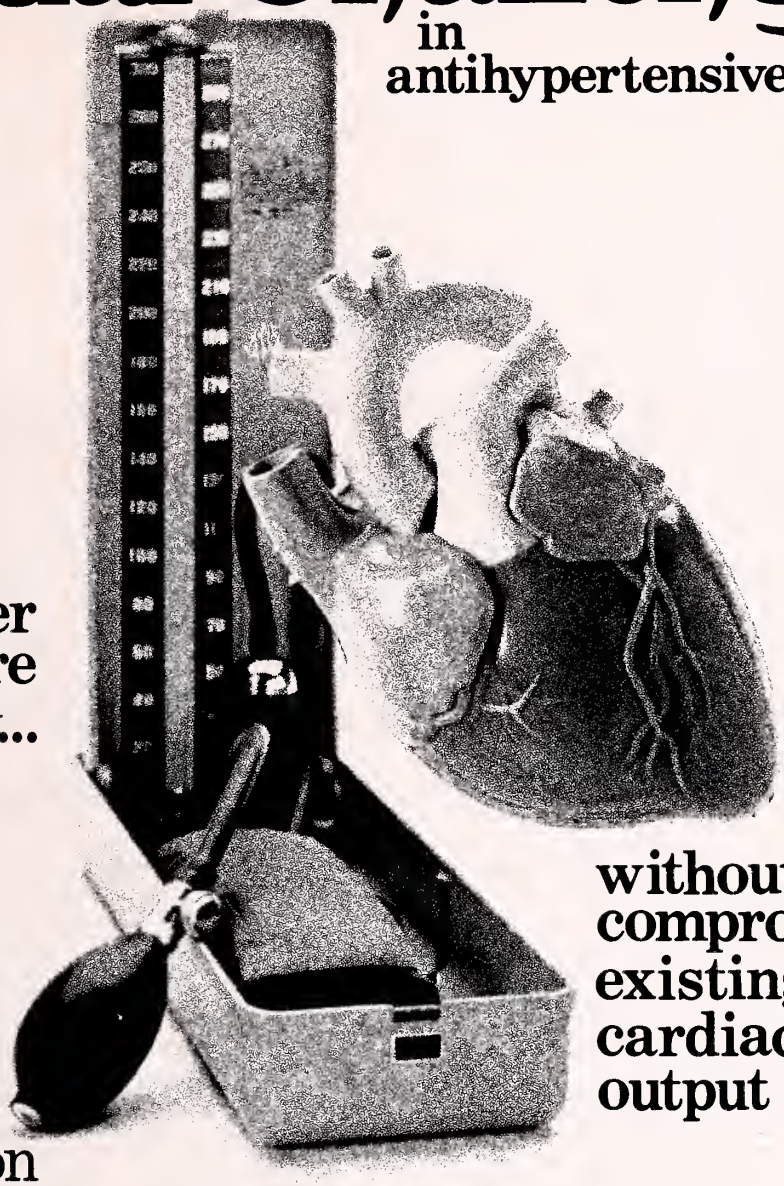

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For a brief summary of prescribing information, please see following page.

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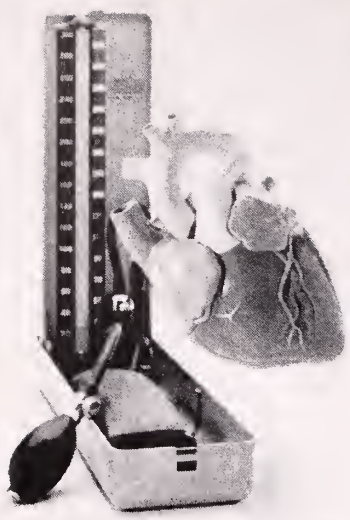

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Contraindications: Active hepatic disease, such as acute hepatitis and active cirrhosis; if previous methyldopa therapy has been associated with liver disorders (see Warnings); hypersensitivity.

Warnings: It is important to recognize that a positive Coombs test, hemolytic anemia, and liver disorders may occur with methyldopa therapy. The rare occurrences of hemolytic anemia or liver disorders could lead to potentially fatal complications unless properly recognized and managed. Read this section carefully to understand these reactions.

With prolonged methyldopa therapy, 10% to 20% of patients develop a positive direct Coombs test, usually between 6 and 12 months of therapy. Lowest incidence is at daily dosage of 1 g or less. This on rare occasions may be associated with hemolytic anemia, which could lead to potentially fatal complications. One cannot predict which patients with a positive direct Coombs test may develop hemolytic anemia. Prior existence or development of a positive direct Coombs test is not in itself a contraindication to use of methyldopa. If a positive Coombs test develops during methyldopa therapy, determine whether hemolytic anemia exists and whether the positive Coombs test may be a problem. For example, in addition to a positive direct Coombs test there is less often a positive indirect Coombs test which may interfere with cross matching of blood.

At the start of methyldopa therapy, it is desirable to do a blood count (hematocrit, hemoglobin, or red cell count) for a baseline or to establish whether there is anemia. Periodic blood counts should be done during therapy to detect hemolytic anemia. It may be useful to do a direct Coombs test before therapy and at 6 and 12 months after the start of therapy. If Coombs-positive hemolytic anemia occurs, the cause may be methyldopa and the drug should be discontinued. Usually the anemia remits promptly. If not, corticosteroids may be given and other causes of anemia should be considered. If the hemolytic anemia is related to methyldopa, the drug should not be reinstituted. When methyldopa causes Coombs positivity alone or with hemolytic anemia, the red cell is usually coated with gamma globulin of the IgG (gamma G) class only. The positive Coombs test may not revert to normal until weeks to months after methyldopa is stopped.

Should the need for transfusion arise in a patient receiving methyldopa, both a direct and an indirect Coombs test should be performed on his blood. In the absence of hemolytic anemia, usually only the direct Coombs test will be positive. A positive direct Coombs test alone will not interfere with typing or cross matching. If the indirect Coombs test is also positive, problems may arise in the major cross match and the assistance of a hematologist or transfusion expert will be needed.

Fever has occurred within first 3 weeks of therapy, sometimes with eosinophilia or abnormalities in liver function tests, such as serum alkaline phosphatase, serum transaminases (SGOT, SGPT), bilirubin, cephalin cholesterol flocculation, prothrombin time, and bromsulphalein retention. Jaundice, with or without fever, may occur, with onset usually in the first 2 to 3 months of therapy. In some patients the findings are consistent with those of cholestasis. Rarely fatal hepatic necrosis has been reported. These hepatic changes may represent hypersensitivity reactions; periodic determination of hepatic function should be done particularly during the first 6 to 12 weeks of therapy or whenever an unexplained fever occurs. If fever and abnormalities in liver function tests or jaundice appear, stop therapy with methyldopa. If caused by methyldopa, the temperature and abnormalities in liver function characteristically have reverted to normal when the drug was discontinued. Methyldopa should not be reinstituted in such patients.

Rarely, a reversible reduction of the white blood cell count with primary effect on granulocytes has been seen. Reversible thrombocytopenia has occurred rarely. When used with other antihypertensive drugs, potentiation of antihypertensive effect may occur. Patients should be followed carefully to detect side reactions or unusual manifestations of drug idiosyncrasy.

Use in Pregnancy: Use of any drug in women who are or may become pregnant requires that anticipated benefits be weighed against possible risks; possibility of fetal injury can not be excluded.

Precautions: Should be used with caution in patients with history of previous liver disease or dysfunction (see Warnings). May interfere with measurement of: uric acid by the phosphotungstate method, creatinine by the alkaline picrate method, and SGOT by colorimetric methods. Since methyldopa causes fluorescence in urine samples at the same wavelengths as catecholamines, falsely high levels of urinary catecholamines may be reported. This will interfere with the diagnosis of pheochromocytoma. It is important to recognize this phenomenon before a patient with a possible pheochromocytoma is subjected to surgery. Methyldopa is not recommended for patients with pheochromocytoma. Urine exposed to air after voiding may darken because of breakdown of methyldopa or its metabolites.

Stop drug if involuntary choreoathetotic movements occur in patients with severe bilateral cerebrovascular disease. Patients may require reduced doses of anesthetics; hypotension occurring during anesthesia usually can be controlled with vasopressors. Hypertension has recurred after dialysis in patients on methyldopa because the drug is removed by this procedure.

Adverse Reactions: *Central nervous system:* Sedation, headache, asthenia or weakness, usually early and transient; dizziness, lightheadedness, symptoms of cerebrovascular insufficiency, paresthesias, parkinsonism, Bell's palsy, decreased mental acuity, involuntary choreoathetotic movements; psychic disturbances, including nightmares and reversible mild psychoses or depression.

Cardiovascular: Bradycardia, aggravation of angina pectoris. Orthostatic hypotension (decrease daily dosage). Edema (and weight gain) usually relieved by use of a diuretic. (Discontinue methyldopa if edema progresses or signs of heart failure appear.)

Gastrointestinal: Nausea, vomiting, distention, constipation, flatus, diarrhea, mild dryness of mouth, sore or "black" tongue, pancreatitis, sialadenitis.

Hepatic: Abnormal liver function tests, jaundice, liver disorders.

Hematologic: Positive Coombs test, hemolytic anemia. Leukopenia, granulocytopenia, thrombocytopenia.

Allergic: Drug-related fever, myocarditis.

Other: Nasal stuffiness, rise in BUN, breast enlargement, gynecomastia, lactation, impotence, decreased libido, dermatologic reactions including eczema and lichenoid eruptions, mild arthralgia, myalgia.

Note: Initial adult dosage should be limited to 500 mg daily when given with antihypertensives other than thiazides. Tolerance may occur, usually between second and third month of therapy; increased dosage or adding a thiazide frequently restores effective control. Patients with impaired renal function may respond to smaller doses. Syncope in older patients may be related to increased sensitivity and advanced arteriosclerotic vascular disease; this may be avoided by lower doses.

How Supplied: Tablets, containing 125 mg methyldopa each, in bottles of 100; Tablets, containing 250 mg methyldopa each, in single-unit packages of 100 and bottles of 100 and 1000; Tablets, containing 500 mg methyldopa each, in single-unit packages of 100 and bottles of 100.

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National health insurance proposals approach the problem of cost controls after the fact, that is after the service has been rendered. All solutions are attempts to control a fiscal problem. None address the need for a national medical policy.

Cost Control Before the Fact

The purpose of insurance is to spread the risk in an equitable way. It seems to me that it should impose a user responsibility, a variable deductable with the availability of a government guaranteed post-payment loan (if needed). Under any national comprehensive health insurance, no one will be deprived of care because of inability to pay or to suffer financial hardship because of a catastrophic, unpredictable illness. However, national comprehensive health insurance with its detailed controls may not prevent rising costs. Experience in Canada, Sweden, and England so far demonstrate that health costs continue to rise very rapidly.

The present burden upon providers to control utilization is of limited success. It seems to me without penalizing either the elderly or the poor that the variable deductable will put responsibility upon the user. All families would be in the decision making process; none would be deprived of access to medical care. All families would be exposed to a limited fixed premium. Cost conscienceness would be fairly shared between providers and patients. This should be an acceptable proposal to physicians and insurance companies, hospitals would begin to return to a freedom in fiscal matters that they have not had since Medicare laws were enacted. If this is such a good scheme, why is it politically unpopular? Economists have recognized that his may be the only method in which an incentive for cost control will work.

Colvin H. Agnew, M.D.,
President,
Montana Medical
Association

The rising cost of medical care is a serious problem. To call it a crisis is probably justified, even in this world of unending crises. Exploitation of crisis is common in our society and in our political process. Exploitation is not necessarily bad. However, because of expediency, irresponsibility, or bias, leadership does not always recognize it.

Crisis and Exploitation

The severity of the increase in cost of medical care has been exaggerated. The cost has increased rapidly over the past few years, but so has the cost of everything else. In five years, from 1970, medical costs increased ten per cent less than food costs and only two per cent more than the increase in income of the intermediate family. The Consumer Price Index shows a rise of physicians' fees from 100 to 192.3 over the past ten years compared to an increase to 183.2 for all services - not the often repeated "twice as fast as the cost of living," a statistic derived from a period during the catch-up phase after the freeze on medical services was terminated. Although an increase in the GNP is usually considered a success

for the general economy, the increase from 7.2 to 8.6 for medical care is viewed as alarming. Unless the increase in cost of medical care is presented in perspective-namely, as primarily a reflection of the general inflation - there will be an unattainable public expectation which will block intelligent solution of the problem.

The crisis in cost of care is exploited to promote legislation in every field of health care. The legislative proposals of each of the many nonphysician provider groups desirous of expanding their field of activity are advanced as mechanisms to reduce costs. The argument that services by these groups is less expensive cannot be substantiated, even without consideration of quality of medical treatment. Like the severity of the rise in cost of medical care, in any health care debate, the argument that an aim of a specific proposal is cost containment usually is unchallenged.

The issue of rapidly rising medical costs is, of course, readily capitalized upon by those dedicated to a radical change of our health delivery system. The issue is used to promote rate setting and ceilings on hospital charges without consideration of the fact that most of the increase in costs of hospital care is caused by continuing inflation. In the crisis atmosphere of "exploding" cost of medical care, the issue of the CT Scanner, a major scientific breakthrough of our lifetime, is rarely understood by the public or objectively debated by the politician or legislator.

Most physicians are aware of the rising cost of medical care. Many are committed to all-out effort in cost containment. Physicians appreciate that developing technology will cause the health care industry to grow at a rate significantly greater than the economy as a whole. The rising cost of medical care is a perplexing and complex problem, but the issue should not be exaggerated and it should not be exploited in promotion of changes in the health care delivery system.

On Death

I've never been able to find the slightest bit of humor relating to death. About some other tragic matters, perhaps, but not about this morbid issue. That is never until I read that "Legislation to establish a legal definition of death has been introduced in the New Jersey legislature — prompted by the legal debate over 21-year-old Karen Quinlan." (American Medical News Feb. 6.) There are now bills in several state legislatures addressing this or related issues.

Some forms of animals have been on this earth for 500 million years. The earliest recorded remains of the species homo sapiens is variously dated from 300,000 to 450,000 years ago, in the middle Pleistocene period. They have all been dying, or been dead for a very long time, or have they? Obviously New Jersey doesn't know, nor has the U.S. Supreme Court provided a definite answer.

In light of our present sophistication in the field of medicine, legislators are dragging their posteriors. Biology biophysics, biochemists, bionuclear medicine, and many other "bios" keep advancing, while pertinent legislations tend to remain as antiquated as the orthodoxy of Catholicism, Judaism and Moslemism. This is true in most states regarding heterologous artificial insemination, organ transplants, the issue at hand, and in numerous other medical areas.

One can understand a lag between progress in science and legislation to deal with the associated problems that arise, but isn't it a bit ludicrous at this stage of our living, having been on this planet so long, not to know when there is no life in a person?

It's long overdue to have the brains of our judicial system conclave with our best bioscientists (and perhaps philosophers, too) and decide once and for all when a person has expired. It's just possible that people who think they are living are really dead (I know a few) and others who are

buried could possible be useful in the legislature.

Sol T. De Lee, M.D.,
Las Vegas, Nevada

To the Editor:

I was absolutely astounded when I read the Letter To The Editor in a recent issue of your Journal written by George B. Markle, IV, MD in which he unfolded an intriguing and strangely interesting story about an old crystal ball he purchased in Carlsbad, which has supposedly been owned by an itinerant Gypsy.

It was like a magical coincidence when I recalled that my father had brought back a similar crystal ball from County Cork, Ireland which he had obtained from a tinker's wagon at the side of the road. It had been stored away in the garage here for over forty years.

I scurried out to the garage and rifled through a lot of old junk: an ice-cream freezer, Christmas decorations, comic books, etc. Finally, Eureka! I found the ball, brought it into the house — washed, scrubbed and polished it to a glistening hue and placed it on the coffee table; the late afternoon sun streaming through and igniting it like a great diamond afire.

It seemed to be almost the same type Doctor Markle had described. He could see the future; could read the stock-market prices, and also see the prognosis for his patients. I thought perhaps I could share the same exceptional advantage. With almost breathtaking, frightening enthusiasm I gazed into the sun-streaked crystal ball and saw a world as it will be a hundred years from now. I was aghast and speechless at what I saw.

It appeared that a hundred years ahead from now society had regressed to an almost feudal-like era, following what was called "The War To End All Wars". There were people living in non-irradiated areas, all seemingly living quite well, healthy and happy. There was no medicine of any kind, in that all the living people on earth were absolutely the survival of the fittest. There was no sickness, except for a refractory syphillis that still had persisted from the Twentieth Century. There were no doctors, because long ago they had started an automatic built-in self-destruct system, so now they were all working as undertakers.

There was no possibility of a war since there were no weapons left; also, people did not know what to fight about. They seemed almost totally preoccupied with love-making. An important difference was the complete absence of any form of government, since there were no cities, no great big buildings, no Military, no bureaucratic organizations and nobody could be bothered listening to anybody about anything.

People had learned to live on the land, till the soil and raise their animals. They lived in cozy cabins and warm caves; cut wood, went hiking, hunting, fishing, and had babies — to everyone's delight.

There was essentially no formal education because people knew only too well what that had done. Everyone lived in a very simple Eutopian atmosphere, developing the natural resources. They passed their information from mouth-to-mouth, with folk-lore stories, Lawrence Welk songs and dances over to the next generation. People traveled by various beasts and boats from place to place to visit — eating, imbibing, swimming, playing games and telling funny stories of the old times when people acted like automatic ice machines. Entertainment consisted mostly of finding and reading old scripts from W.C. Fields and Milton Berle. Along the roadways you could see vestiges of the archaic civilizations such as automobile CB's, calculators, an air-conditioning compressor or a piece of a fusion reactor. Many of these artifacts often were hung in the great rooms of the caves as ornaments over the fireplace.

However, there were a few die-hard psychiatrists left. Although people were very healthy, they still got up-tight on occasion. But

most psychiatrists would leave their crystal ball on the desk and lie on their couch. When the patient came in, the doctor would say, "Just leave the fee and look into the crystal ball". This made everybody happy because the patient really didn't have to tell anything and the Doc didn't have to listen to anything — he just had a good snooze.

Now, with my crystal ball, I simply leave it on my desk in the office for the patient's use. I generally stay home sunning myself at poolside, sipping Gatorade, and let the crystal ball do all the work. On rare occasions I phone the office to check on my secretary and back taxes. It has been a tremendous asset to me and allows me much time to enjoy life.

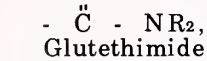
I am eagerly looking forward to meeting with Doctor Markle and getting our glass balls together, as we have foretold the future in such totally diametrically-opposite directions. Since two balls are better than one, I am sure that between us the real solution lies somewhere in the middle of all this.

John F. McCarthy, VIII, MD
Albuquerque

To The Editor:

In the January-February 1977 issue of the Rocky Mountain Medical Journal an article titled "Glutethamide Poisoning" appeared which required clarification and correction. First, there is no such drug as glutethamide. The authors probably mea lutethimide (DoridenR). The use of imide is unacceptable. There is a significant difference between amide and an imide. An imide has the atomic array

; the amide has the array



where R=H, alkyl or aryl. Glutethimide has a molecular structure possessing the imide group. Therefore, including imide in the name glutethimide has a definite significance and any reference to the drug being an amide is erroneous.

Accepted use of abbreviations should be reviewed by the authors. The use of the abbreviation Hg for the glutethimide metabolite is misleading. Hg is the symbol for the element mercury; use of Hg for anything other than mercury must be avoided. The way the authors present the abbreviation is confusing: 4Hg (4-hydroxy-2-ethyl-2-phenylglutarimide) conveys the idea that the metabolite is a mercury complex; and mercury is used as a location reagent in the thin layer chromatographic analysis for glutethimide and its metabolites. The use of Hg as an abbreviation is misleading and confusing.

The third and most significant error in the article is their reporting of the drug levels. The authors report blood glutethimide levels reaching 35 mg /ml. This is absurd. A level of 35 mg /ml is the same as 3500 mg /dl or 35000 ug /ml. Obviously the authors intended to use microgram (ug) instead of milligram (mg). But one can't be sure because in the section under Comment, they state the toxic range of glutethimide is 10-80 mg /ml. Winek,[1] a noted authority on drug levels, gives the toxic range of glutethimide as 10-80 mg /liter and not 10-80 mg /ml (a thousand fold difference). The authors should pay more attention to units.

There is one interesting aspect of glutethimide poisoning that the authors failed to note: this case demonstrates the glutethimide "rebound"[2] phenomenon, i.e., the blood level suddenly rises after steadily declining. The data in Table 1 of their article indicate the glutethimide level jumped from 1.1 (whatever the units) to 4 between 11:00 A.M. and 11:00 P.M. on 1-14-76. Till that time the level steadily declined from 35. Generally the condition of the patient deteriorates during this "rebound" period. If in fact this is a true case of the "rebound" phenomenon, it can be argued that the importance the authors place on the concentration of the metabolite is not correct. The level of the metabolite should also have increased following the increase in the level of the parent glutethimide. No increase is reported.

The authors do present an interesting case but should concentrate on specific details. The literature is confusing enough with respect to therapeutic, toxic and lethal drug blood levels.

Sincerely,
R.W. URICH, M.D.
Colorado Springs, Colorado

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Unfortunately the corrected proofs were received from Dr. Galloway after the article had gone to press. The corrections were essentially those indicated by Dr. Urich. The Editor.

Office tests of endocrine function

W.A. Reynolds, MD, Missoula, Montana

Many basic tests of endocrine function can now be performed as office procedures, including a new growth hormone stimulation test.

Assessment of endocrine function has undergone drastic change in recent years with the development of many hormanal assays, particularly radioimmunoassays. Most of these are available to us in practice, as marketing of reliable immunoassay kits has enabled our local laboratories to perform many of these tests. Nearly all the assays are readily available by using the excellent commercial reference laboratories, such as Bio-Science and the Mayo Clinic.

I have been particularly interested in the office use of these tests, avoiding costly hospitalizations for diagnostic workups when possible. This paper will describe some new and some not so new procedures and tests which any physician can perform in his office in the evaluation of endocrine status.

Human Growth Hormone (HGH) Assay

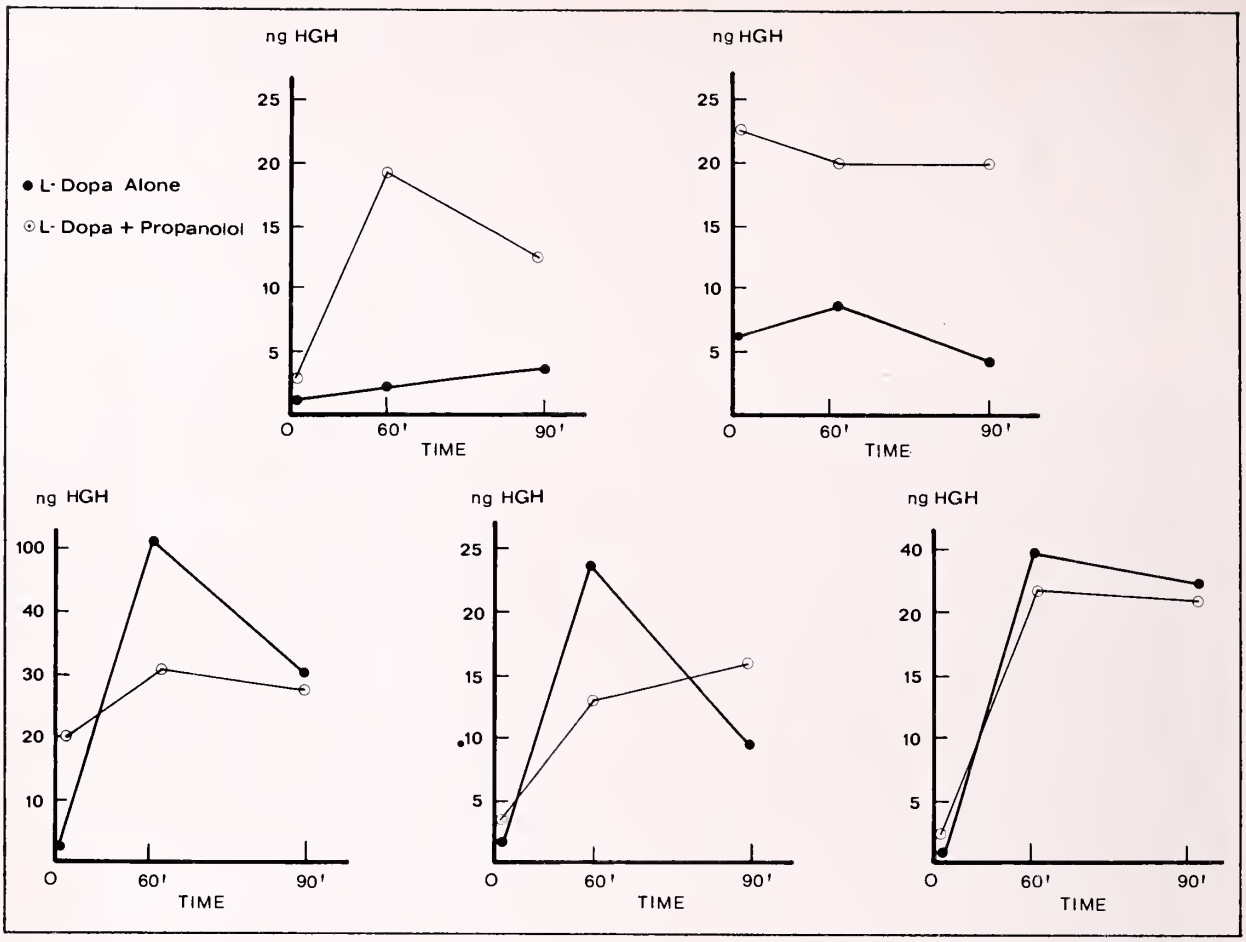
Clinical suspicion of acromegaly can be confirmed easily by demonstration of nonsupression of growth hormone in venous blood after an oral glucose load. A fasting sample is obtained followed by a standard glucose tolerance test, drawing only the one hour specimen. Plasma glucose and growth hormone determinations are made. Normally, HGH virtually disappears by one hour (2.5 ng-ml or less).[1]

Another obvious use of this assay is in the evaluation of growth failure and short stature, particularly during childhood and adolescence. Since low fasting levels may occur normally, diagnosis depends on failure to demonstrate a rise after appropriate stimulation.

The value of HGH assay is not limited to evaluation of acromegaly and growth failure. Recently, Nelson et al[1] have confirmed previous reports that abnormal HGH secretion is the most frequent endocrine dysfunction among patients with pituitary disease. Of 47 consecutive patients with pituitary disease, nine (acromegalics) had hypersecretion and 29 had HGH deficiency. FSH-LH, TSH, and ACTH were also assayed after appropriate maneuvers. Deficiencies or excesses of these hormones were present in only two patients who did not have HGH abnormality also. It is clear that the best screening test for suspected disease or lesion of the pituitary is an HGH stimulation test.

The classical method of HGH stimulation is to perform an insulin tolerance test causing hypoglycemia which results in HGH release. Because of the hazards of hypoglycemia, this test is not convenient to do safely in an office practice. Other stimulation tests described include infusion of ACTH or arginine, injection of vasopressin or glucagon or pyrogen, ingestion of L-Dopa or tyramepone, and exercise and sleep.

A number of reports comparing these tests suggest that L-Dopa is superior to all but the insulin tolerance test.[2-4] One study of twenty normal adults, reported that 95 percent responded to L-Dopa and only 90 per cent responded to insulin.[4] Eight other studies sup. 2 [2,3,6-10] in the literature



indicate that responses to L-Dopa in children as well as in adults are quite variable In these eight studies, L-Dopa failed in from 5 to 40 per cent of the persons that were responders to the insulin test. Especially prone to failure with L-Dopa were obese persons and children of short stature who were not growth hormone deficient.[11] In 1974 Camanni[12] reported potentiation of the L-Dopa test by giving intravenous propranolol. Peak response occurred between 30 and 90 minutes after giving the drugs. Criteria for an HGH response to any of the above tests varies in different laboratories. A concentration of at least 5 ng-ml is a minimum criterion.[2] Other investigators require a rise of at least 5 ng,[4 10] 6 ng[3] or 7 ng-ml.[1]

L-Dopa works by increasing dopamine levels in the hypothalamus while propranolol makes the alpha adrenergic system more active by inhibiting the beta adrenergic system, both augmenting release of growth hormone.[11] To simplify the L-Dopa intravenous propranolol test, I made a modification by giving 500 mg L-Dopa with 40 mg propranolol orally. Results on five normal young adult laboratory technicians are shown in Fig. 1 comparing L-Dopa alone to L-Dopa plus propranolol. Two subjects failed to respond to L-Dopa; one of these responded to the combined test and the other had high initial level negating evaluation of a response. Three subjects responded to both tests.

Using L-Dopa plus propranolol, eleven patients had normal HGH responses. Patients tested included two with diabetes insipidus, three with amenorrhea, two with short stature, one with an enlarged sella on x-ray, one patient who had secondary hypothyroidism and a normal sella, one patient with suspected Jakob-

Creutzfeldt disease, and one with ACTH deficiency, described briefly in the following section. Four additional patients were of particular interest.

CASE REPORTS

Case: 1 A 40-year-old hysterectomized female had clinical hypothryoidism documented by T4 and T3 uptake and had a low TSH. She had no visual field defects or abnormality of the sella on x-ray. L-Dopa-propranolol orally produced no response of growth hormone; however, her fasting blood sugar the day of the test was 140 mgper cent. Repeat testing using standard intravenous insulin test produced a normal response with blood sugar reaching 50 mg per cent at 30 minutes, the growth hormone rising to 12.3 mg-ml at 60 minutes. The elevated blood sugar may have blocked the response to the L-Dopa-propranolol test.

Case 2: A sixty-year-old adult male presented with complaints of fatigue and decreased pontentia. He was found to have low T4 and a low TSH and was thought to be hypothyroid. However, subsequent testing. revealed a normal T3 uptake and a normal thyroid binding globulin assay; pituitary gonadotropins, serum prolactin, and testosterone determination were normal. This man failed to respond to L-Dopa alone and subsequently to L-Dopa-propranolol. However, following three months of tricyclic therapy for depression, he was retested with L-Dopa-propranolol and had a normal response with growth hormone reaching 7.2 ng-ml.

Case 3: A 19-year-old female with secondary amenorrhea was found to have a unilateral inferior temporal visual field defect. Her serum prolactin was elevated at 40 ng-ml. She had an equivocal growth hormone response to L-Dopa-propranolol. Maximum HGH level obtained was 6.2 ng-ml. Subsequent polytomography of the sella, Actascan (Computerized Axial Tomography), pneumoencephalography, and cerebral arteriograms at the University of Minnesota were all normal. No definite diagnosis has been established.

Case 4: A patient with Cushing's disease was tested preoperatively for possible pituitary adenoma. The L-Dopa-propranolol test gave a normal HGH response of 14.5 ng-ml at 60 minutes and 1.6 ng-ml at 90 minutes. This was followed immediately by an IV insulin test with plasma glucose levels falling to 40 ng-ml but with no HGH response (values were 1.6, 1.9, 2.4 and 3.1 ng-ml during the 90 minute test).

While we were doing these studies, Collu et al reported on their results also using oral propranolol plus L-Dopa in comparable doses to those used in my series. There were no false negatives in seventeen patients of short stature. At present, I would recommend the L-Dopa plus propranolol method as the first test to be used. Only nonresponders should be retested by insulin tolerance. The only untoward effect is nausea in some patients.

Adrenal Function Tests

Cushing's disease is occasionally suspected but rarely found. The obese, mildly diabetic, hypertensive female with abdominal stria is not a rarity in medical practice. A simple screening test to rule out Cushing's syndrome is most helpful. The over night Dexamethasone suppression test is the simplest answer. One milligram of Dexamethasone is given at 11 pm, and the following morning at 8 am blood for plasma cortisol is drawn. A barbiturate given at bedtime to insure sleep is a worthwhile addition. Normal persons suppress to 2 μg-100 ml or less. In Cushing's syndrome values are above 10 μg-100 ml. Values may range up to 10 μg-100 with stress, obesity, depression, and estrogen therapy.

A simple office procedure for evaluating adrenal cortical responsiveness is the cosyntropin (Cortrosyn) ACTH test.[14] Cosyntropin is a synthetic short chain ACTH analogue which carries less risk of allergic reaction than other ACTH preparations. Baseline plasma cortisol is determined and repeated 30 to 60 minutes after giving 250 μg of intramuscular or intravenous cosyntropin. An increment of greater than 6 μg-100 ml and a concentration of more than 20 μg-100 of cortisol is a normal response.

Evalution of pituitary-adrenal axis indirectly testing ACTH responsiveness is usually performed in the hospital using an oral or intravenous metyrapone (metopirone) schedule requiring up to 72 hours to complete. A shortened version for outpatients is sometimes useful.[15] Thirty mg /kg of body weight of metyrapone is given at 11 pm with food to reduce gastric irritation. At 8 am the next morning venous blood is drawn for plasma 11-doxycortisol. Values exceeding 7 μg-ml are considered normal. We have used this test in several patients with a

normal response. One patient who had a positive test gave a history of two severe episodes of shock, closely following major surgery. In both instances, the patient responded to intravenous hydrocortisone. Adrenal responsiveness to the Cortrosyn test was normal. No other evidence of pituitary dysfunction or insufficiency was found.

Each of the above tests should be considered as screening tests. If abnormal or borderline results are obtained, the appropriate standard tests requiring hospitalization should be considered.

Thyroid Function Tests

Recent additions to laboratory assessment of thyroid function include the thyroid stimulating hormone (TSH) assay, the assay of plasma triiodothyronine (T3), and the thyrotropin releasing hormone (TRH) stimulation test. TSH should be determined in all patients suspected of being hypothyroid when etiology of the hypothyroidism is not apparent. In primary hypothyroidism the value is always elevated and elevation preceeds significant changes in T3 and T4 values. Elevated TSH thus confirms the diagnosis of primary hypothyroidism. If the TSH is low, disease of the hypothalamic-pituitary axis is the cause. Following the TSH values to the normal range is also helpful in evaluating the adequacy of thyroid replacement therapy.[16]

Triiodothyronine (T3) assay, not to be confused with the T3 uptake test, may confirm the presence of hyperthyroidism when the T4 and T3 tests are normal (T3 toxicosis).[17] Radioactive iodine uptake in these cases may be normal or elevated. T3 is also disproportionately increased in respect to T4 in treated hyperthyroidism, early thyroid failure, and iodine deficiency goiter.[18] TRH has been synthesized and can be administered intravenously. A dose of 500 µg of TRH is given in 15 to 30 seconds intravenously. Comparing the baseline TSH level to the post injection (within 20-40 minutes) TSH level is a very sensitive test of thyrotoxicosis of any etiology. Normally a sharp rise of TSH occurs promptly. The TSH response is greatly diminished in thyrotoxicosis, in patients with functioning thyroid adenomas who are euthyroid and in one third of patients with euthyroid Grave's disease. TSH response is exaggerated in hypothyroidism even when the deficiency is slight and serum TSH concentrations are only marginally increased. TRH has recently been released in this country for general use. This test may replace the triiodothyronine thyroid suppression test as the initial procedure in evaluation of euthyroid Grave's disease when exophthalmos is the presenting complaint.[18,19]

Renin Assay in Hypertension

After induction of volume depletion, about twenty per cent of the hypertensive population will have a low plasma renin. This identifies a group of patients that will respond to spironolactone (Aldactone) as well as usual diuretic therapy. Very low renin values also suggest aldosteronism and coupled with hypokalemia identify the patient for further study. Finally, about ten per cent of essential hypertensives can be identified in a high renin group. These patients should be carefully evaluated for renovascular hypertension. Manipulations to stimulate renin usually preceed the renin determinations.

Standard procedures commonly used include a diet of 10mEq of sodium for five days in the hospital plus three hours of upright posture or three doses of furosemide orally at six hour intervals as described by Carey. Laraugh popularized a procedure comparing 24 hour sodium excretion to plasma renin in the ambulatory subject.[21,23] Recently two simplified furosemide stimulating tests have been reported. Two weeks without antihypertensive therapy, two months without oral contraceptives and a normal sodium diet preceed the tests. Kaplan et al[22] evaluated the plasma renin drawn 30 minutes after giving 40 mg of furosemide intravenously, with the patient remaining upright and found close correlation with the plasma renin drawn after two hours of upright

posture preceeded by three days of low salt diet. Low renin values were 1.0 ng / ml /hr for hour or less for whites and 0.5 ng /ml /hr or less for blacks. High values were greater than 5 ng /ml /hr. Wallach et al[21] modified the test giving 60 mg furosemide orally followed by 5 hours of upright posture. There was a 93 per cent correlation of this procedure compared with the hospital salt depletion protocol. Values for low renin hypertension were less than 1.5 ng /ml /hr. Three patients with renovascular hypertension had renin values of 7.7 ng /ml /hr or higher. Few clinicians feel that renin classification of all hypertensives is practical or cost effective, but an easy relatively inexpensive office method makes it a feasible and valuable procedure in helping to identify cases of reversible secondary hypertension that make up ten per cent of the total hypertensive population.

Summary

There are presently many tests for assessment of endocrine function which can be conducted in the office. Growth hormone assay, adrenal function tests, thyroid function assays, and renin assay in hypertension are presented and evaluated. A new oral growth hormone stimulation test is described.

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Ehlers - Danlos syndrome with spontaneous rupture of the esophagus

Report of First Case

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The Ehlers-Danlos Syndrome is a familial disorder of connective tissue.[8] An accumulating literature attests to the surgical significance of this condition which has been associated with spontaneous rupture of large blood vessels[2, 4, 6] and portions of the intestinal tract,[1-3, 5, 7] hernias, joint subluxations, gastrointestinal bleeding, ocular abnormalities, and a variety of other complications requiring surgical treatment[1 9]. Our recent experience with spontaneous esophageal rupture in a patient with Ehlers-Danlos Syndrome appears to be unique. Although spontaneous rupture of the colon and small intestine have been recorded in patients afflicted with the Ehlers-Danlos Syndrome, esophageal rupture in association with this condition has not been previously reported.

CASE REPORT

A 37-year-old white man was referred with the diagnosis of spontaneous esophageal rupture in May 1974. Approximately 12 hours prior to admission he had awakened nauseated, and he had vomited forcefully on several occasions. During one of the vomiting episodes he had experienced sudden, severe pain in the epigastrium, left chest, and left shoulder. Shortly thereafter he was admitted to a local hospital where a chest roentgenogram was made, a nasogastric tube was inserted, parenteral narcotics and antibiotics were administered, and he was transferred by ambulance.

Diagnosis of Ehlers-Danlos Syndrome had been made for this patient at age 6 months. He had had multiple minor injuries. By the time he was 14 years old he had estimated that he had over 400 sutures for a variety of lacerations everywhere on his body. In earlier life he had had troublesome bleeding, but in recent years his blood clotted satisfactorily. He had had dislocations of both shoulders. In 1970 he had had surgical drainage of a left temporal lobe abscess due to Nocardia asteroides. The brain abscess had resulted in seizures in the past, but he had had none since 1972. Daily, he took Dilantin, sufadiazine, and tetracycline.

The Patient's father had had Ehlers-Danlos Syndrome with mild symptoms, and had died of gastrointestinal hemorrhage. His mother was living but had "arthritis". A sister had died at an early age of "pneumonia", and a brother apparently was well. The patient had a son age five years who was said to be healthy.

Examination on admission revealed a critically ill man who appeared older than his chronologic age. He was uncomfortable and restless, and brachial blood pressure was 96 /60 mm Hg. His pulse rate was 130 per minute. Respirations were labored and 34 per minute. There was remarkable hyperelasticity of his skin and hyperextensibility of many of his joints. Surgical scars were noted in the left temporal area, and there were multiple papyraceous scars on his body, especially on the legs, feet and hands where there were also multiple, discolored pseudotumors and deformities of his fingers and toes. Subcutaneous emphysema was obvious in the left side of his neck and upper left chest. There was tachycardia but the heart rhythm was regular, and no murmurs were detectable. Breath sounds were decreased in intensity over the left chest posteriorly and laterally. Moderate abdominal tenderness was elicited with marked guarding in the left upper quadrant. No bowel sounds were audible. There was no inguinal hernia.

Laboratory studies soon after admission showed slightly elevated values for blood hemoglobin and hematocrit. Leucocyte count was normal with a moderate left shift in the differential. Blood group was A Rh positive. Routine urinalysis was negative. Prothrombin activity was 100 per cent. Partial thromboplastin time was reported to be 44 seconds (N=25-40), bleeding time two minutes and clot retraction had not begun at one hour. Blood platelets were 120,000 per cubic milliliter, and blood fibrinogen was 320 milligrams per deciliter (N=170-410). Roentgenogram of the chest showed pneumomediastinum and left hydropneumothorax.

Emergency operation was undertaken soon after admission. The left pleural space was explored through the bed of the nonresected ninth rib postero-

laterally. There was approximately a liter of cloudy, gray pleural fluid from which neisseria, alpha hemolytic streptococci, and candida albicans were subsequently isolated. Patchy areas of necrosis were noted in the parietal pleura. The pulmonary ligament was edematous and friable, and there was a perforation in the mediastinal pleura over the lower esophagus. A small sliding esophageal hiatal hernia was demonstrable. Just proximal to the esophagogastric junction there was a longitudinal 2.5 centimeter laceration of the esophagus. The mucosa at the site of the laceration was edematous, but there was no detectable hematoma or ecchymosis in the esophageal wall.

The laceration was readily repaired in two layers with Dexon sutures. Bleeding was not troublesome nor were the tissues of the esophagus unusually friable. The sliding hiatal hernia was not repaired. At the conclusion of the procedure the pleural space was irrigated with large quantities of saline, and a solution of keflin containing saline was instilled. Two pleural catheters were placed for temporary drainage. The tissues of the chest wall, including musculature, fascia, and skin were noticeably more elastic than usual, but closure with Dexon and chromic catgut sutures was accomplished without difficulty, there being no tendency for the sutures to tear the tissues or to pull out. No bleeding problem was encountered.

Nasogastric suction and closed pleural catheter drainage were continued for four days after which the tubes were removed. Keflin and cleocin were administered intravenously during the first four postoperative days. Except for a small, culturally sterile hemopneumothorax requiring aspiration on the seventh day, the postoperative course was uncomplicated. At the time of dismissal from the hospital on the eleventh day, his gastrointestinal tract was functioning normally, and the thoracic incision was healing satisfactorily. He had no dysphagia, nor had he, at any time in the past experienced symptoms of esophageal hiatal hernia or esophagitis. Upper gastrointestinal x-rays with barium several weeks later showed no abnormalities, the esophageal hiatal hernia not being demonstrable.

Discussion

McCusick,[9] in 1974, noted that seven distinct types of Ehlers-Danlos Syndrome had been identified. The first four types have distinguishing clinical characteristics, and types one through three are inherited as autosomal dominants. Type five has a sex-linked recessive inheritance, and types six and seven are classifiable on the basis of biochemical abnormalities of collagen tissue. The most common manifestations of the Ehlers-Danlos Syndrome are those involving the skin and joints. Classically the joints are hyperextensible, and the skin is excessively stretchable and fragile.

Visceral complications may occur in several forms of the disease, but McKusick states that spontaneous rupture of the blood vessels and the intestinal tract is particularly a characteristic of type four. This type is designated the arterial or ecchymotic variety. McKusick has indicated there was preliminary evidence for a defect in the synthesis of vascular collagen in this form of the Ehlers-Danlos Syndrome.

The patient herein reported was not a single, isolated case in his family; his father was said to have had Ehlers-Danlos Syndrome. This apparently is not consistent with his being classified as a type four variety. In addition, there was no evidence at operation of hemorrhage as the initiating factor in his esophageal perforation.

It must, of course, be recognized that the association of Ehlers-Danlos Syndrome and spontaneous esophageal rupture in this patient may have been entirely fortuitous. This is unlikely, but certainly

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there were adequate mechanical factors (i.e. the forceful vomiting) to account for the esophageal rupture which occurred in the usual location just above the diaphragm. It is also of interest that no difficulty was encountered in the repair or in the subsequent healing of the esophageal laceration or the thoracic incision.

Summary

A case of spontaneous esophageal rupture in a patient with Ehlers-Danlos Syndrome is reported. The complication was managed by primary, transthoracic repair with a satisfactory result.

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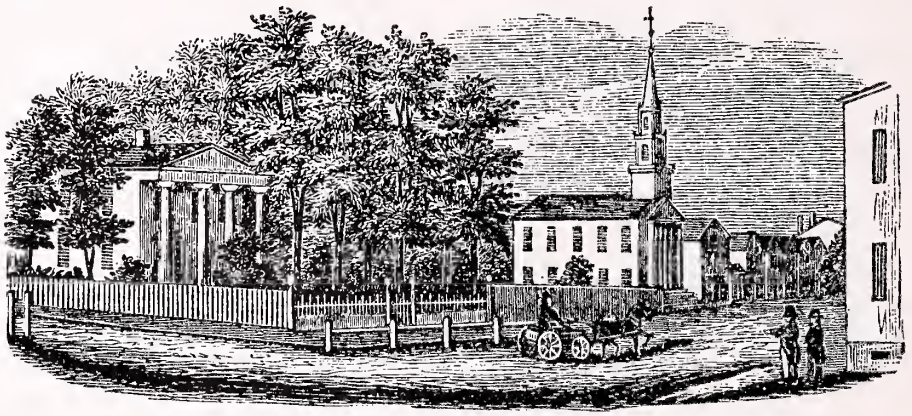

Western view in Dansville, Livingston county.

Complete transection of urethra:

A Urogenital Ski Injury

Robert J. Towers, MD, Bozeman, Montana.

During the past few years we have seen a definite increase in the number of ski injuries to the perineum and genital areas. Shorter, better designed skis, better boots and groomed slopes have allowed skiers to ski under control at much higher speeds than previously. At the same time, markedly improved safety bindings have decreased the chances of lower extremity fractures. Despite an impressive fifty per cent overall decrease in serious fractures, there seems to be an increase in the number of blunt injuries to the upper body including the genital and perineal areas.[1] This is a report of a case of a complete transection of the bulbous urethra which was successfully treated by immediate exploration and primary repair.

CASE REPORT

A 24-year-old novice skier hit a small bump while skiing at a high rate of speed. As he fell into the sitting position, the toe piece of his "step-in safety bindings" released. His ski shot forward but was held to his foot by the safety strap. The ski tip stuck in the snow ahead of him and the butt rammed into his perineum as he slid forward. Following the injury he was able to ski off the hill unassisted but later noticed that his scrotum was markedly swollen and ecchymotic. He was seen eight hours after injury because he was unable to void.

Physical examination showed marked edema and ecchymosis of the scrotum and perineal area. A retrograde-urethrogram showed extensive extravasation of contrast material at the bulbous urethra (Fig. 1). Exploration of the perineum confirmed that the urethra had been completely transected and the proximal and distal ends had retracted and were separated about 5 to 6 cms. The ends of the urethra were debrided and easily sutured together over a 20 French silastic catheter without tension.

Suprapubic cystotomy had been required to find the proximal end of the severed urethra and a large suprapubic mushroom catheter was left in the bladder and placed on drainage. Penrose drains were placed locally and the wounds closed. The patient did well postoperatively. On the 10th postoperative day his urethral catheter was removed and a voiding urethrogram obtained (Fig. 2). There was no evidence of extravasation and the urethra was widely patent at the anastomotic site. After the suprapubic tube was removed the patient was able to void with a good stream. A urethrogram obtained one year postoperatively was normal. The patient has continued to do well and has not required dilatations.

Discussion

The classic approach to complete transection of the bulbosus urethra had been surgical exploration with primary repair.[2] Mr. Richard Turner-Warwick[3] recom-

Fig. 1. Retrograde Urethrogram at time of injury.

Fig. 2. Voiding Urethrogram two weeks following repair.

mends primary repair of these injuries but suggests that the anterior part of the repair be left open. He feels that if this is done, fewer stricture problems will arise. In this case, we felt that the anastomosis could be done so easily without tension that this was unnecessary and would prolong the time the Foley catheter would be required. Dr. Hand[2] writes that if a catheter can be placed across the injury, a perineal exploration need not be done but the catheter should be left in place for an extended period. In this case, however, I was able to pass a small rubber catheter across the defect and into the bladder. Later exploration showed that there was a

Fig. 3. Retrograde Urethrogram one year following repair.

5 cm. separation in the proximal and distal urethra. I would anticipate that nonoperative treatment in this case, would have resulted in severe urethral strictures since it has been shown that severe strictures tend to form in areas where the mucosa has retracted.[4] We, therefore, recommend that if there is extensive extravasation on the retrograde urethrogram, the area should be explored and, if possible, a primary repair done even when a catheter can be passed into the bladder primarily.

Summary

The case of primary repair of a complete transection of the bulbous urethra secondary to a ski injury is presented. Where there is an extensive extravasation, exploration and primary repair of distal urethral injuries is advised, even if a catheter can be passed across the defect. It is also felt that changes in ski equipment and techniques are resulting in a decrease of overall ski injuries but are producing an increase in the number of perineal and genital injuries.

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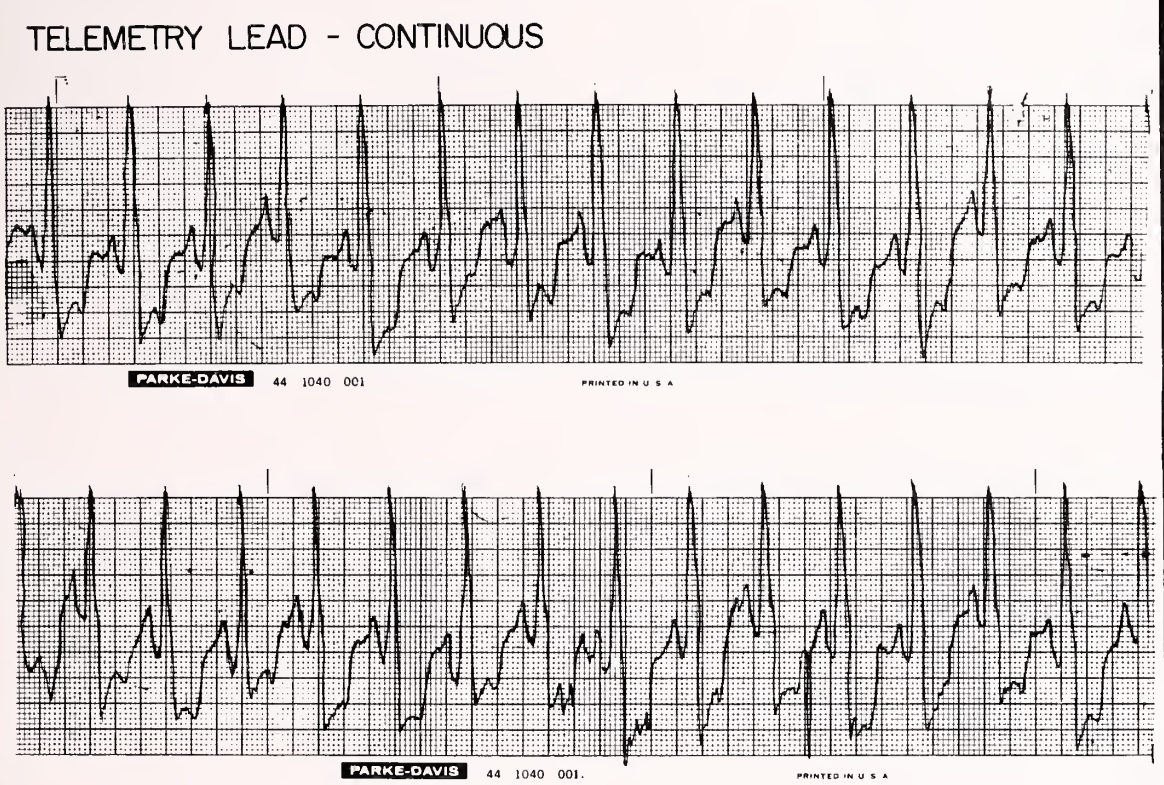
Prepared by Jonathan Abrams, MD, Albuquerque, New Mexico *



This telemetry lead was taken in an emergency rescue vehicle from a 63-year-old female with known coronary artery disease, who was experiencing shortness of breath and chest pain. The cardiac rhythm rapidly degenerated to a slow ventricular dysrhythmia before the patient was successfully treated.

What is this arrhythmia?

See Page 96

* Dr. Abrams is Chief, Division of Cardiology, and an Associate Professor of Medicine, University of New Mexico School of Medicine, Albuquerque.

Intra-aortic balloon assist

Use in a Regional Hospital

H. Dan Adams, MD, W. Stan Wilson, MD, George H. Reed, MD and Peter A. Philips, MD, Missoula, Montana

Remediable myocardial failure is a major therapeutic challenge. Noninvasive measures designed to improve tissue perfusion (pressors and inotropic agents) may accomplish this goal but by doing so increase myocardial work. Thus, they may cause further myocardial deterioration in a failure state. Other regimens which lessen the work of a damaged heart (diuretics and vasodilators) may also result in a lower and potentially deleterious perfusion state. In the late 1960's, a unique invasive device was developed to relieve the failing heart when the above measures had not been successful.[1,2] A sausage-shaped balloon, inflated in diastole by an ECG trigger, was found to be capable of increasing coronary and systemic blood flow if properly positioned in the thoracic aorta. By deflating prior to systolic ejection, the balloon decreased afterload and thus demands on the left ventricle. This combination of effects allowed support of the patient for a period during which transient myocardial damage might be reversed.

Intra-aortic balloon assist (IABA) was initially limited to large university hospitals. However, as sophisticated cardiac care has become available in community centers, IABA has assumed an integral role. A recent survey indicated that one-half of the cardiac surgical programs in the United States utilize some sort of IABA device, and many of the remaining groups plan to institute its use this year.[3] We began our cardiac surgical program with an AVCO IABA unit.† The purpose of this report is to illustrate the use of balloon augmentation in a regional hospital cardiac program.

Technic of Insertion

The technic of insertion has been described in other publications.[4] [5] We have added a few modifications. When cardiac catheterization studies indicate a major increase in operative risk, angiography of the aorto-iliac-femoral runoff is performed. A preferential site for balloon insertion is thus determined. Careful skin preparation is essential to maintain a sterile balloon catheter incision for a maximum of five to seven days. In an emergency situation local anesthesia with 1% Xlyocaine is employed. In the operating room Ketamine anesthesia (0.5mg /kg) is used for the IABA insertion immediately prior to the cardiac operation. The largest balloon that will pass into the thoracic aorta is employed. Occasionally the use of a no. 6 Fogarty catheter for iliac sizing is of aid. An indwelling arterial line and a Swan-Ganz catheter in the pulmonary artery are necessary for continued maximal monitoring of the cardiopulmonary status. The IABA balloon is advanced up the femoral artery until the tip rests just below the left subclavian artery (Fig. 1). Balloon augmentation is begun. A sleeve of 10mm diameter Johnson & Johnson bovine carotid artegraft is sewn to the common femoral arteriotomy to create a side arm for the entrance of the balloon catheter (Fig. 2). We have found a

* From the Cardiac Surgical Program, St. Patrick Hospital, Missoula, Montana

† AVCO Medical Products, Everett, Massachusetts. Model No. IABP 7.

Fig. 1. X-Ray With Balloon In Place.

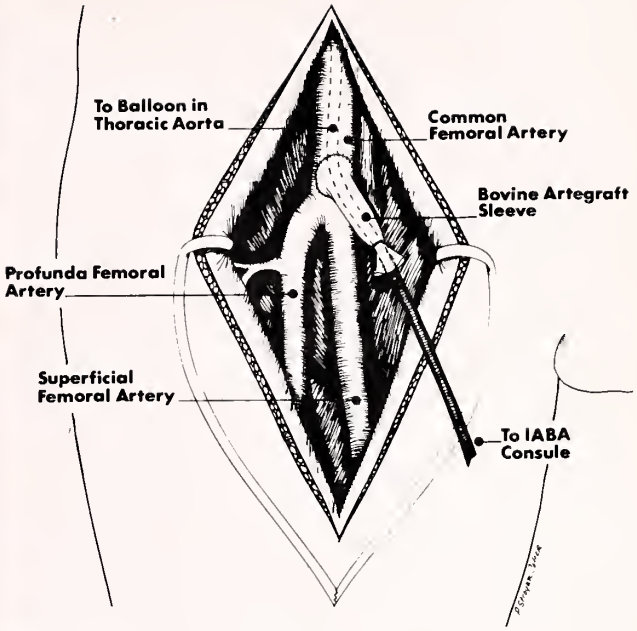


Fig. 2. Illustration of Insertion.

2 to 3cms segment of this material superior to the Dacron cuffs formerly used. The bovine artegraft is soft, supple, excellent for suturing, and immediately hemostatic — a definite advantage during full heparinization for cardiopulmonary bypass. Other means of aiding the myocardium, such as nitroprusside, may be used with potentiated effect when they are employed in conjunction with balloon augmentation.

Antibiotic coverage (usually Ancef), daily changes of the occlusive dressing, and repeated Betadine washings maintain cleanliness of the incision. To date there have been no incisional infections. After a period of two to six days with patient stabilization, the balloon is removed under local anesthesia. We have not had any long-term balloon dependent patients. After balloon removal, a Fogarty catheter is passed into all major branches of the femoral artery to insure good flow. The base of the artegraft is used to close the common femoral arteriotomy.

Indications for Use

Only when all other noninvasive measures have failed, do we institute balloon augmentation for acute myocardial embarrassment (Table I). On five occasions,

TABLE 1

Parameters defining cardiogenic shock

1. Systolic pressure 80mmHg or below
2. Left atrial pressure greater than 20-25mmHg
3. Low urinary output, 20cc or less per hour.
4. Diaphoresis, wet, clammy skin.
5. Confusion and/or obtundation.

we have employed the device to assist patients in deep cardiogenic shock secondary to acute myocardial infarct (MI). On one occasion we have attempted to preserve myocardium in a setting of persistent angina following an infarction before shock could develop. Our most frequent use of the assist device (six cases) has been immediately prior to a cardiac surgical procedure in patients with high risk. Preoperative augmentation can prevent disastrous hypotension and myocardial damage during the operation. On two occasions, when patients were unable to be weaned from cardiopulmonary bypass due to profound myocardial dysfunction, balloon augmentation was initiated. During our first year of cardiac surgery, the IABA device was employed in a total of twelve patients with varying success (Table 2). The following cases illustrate its use.

TABLE 2

Use of IABA, St. Patrick Hospital, Missoula, Montana

Patient	Age/Sex	Indications for use	Length of augmentation	Result
JJ	50/M	Acute cardiogenic shock	4 days	Died 4 months later
LA	50/M	Persistent pain post-MI	3 days	Died in CCU
BT	52/M	Acute cardiogenic shock	3 days	Died 4 months later
JH	65/M	80% left main lesion/CABx5	2 days	Alive and well
JC	33/M	90% left main lesion/CABx3	2 days	Alive and well
RG	67/M	High risk MVR, tricuspid annuloplasty, CABx2	4 days	Alive and well
VJ	47/F	Inability to wean off bypass post-CABx2	5 days	Alive and well
HR	67/M	Multiple total occlusions pre-MI/CABx5/ pending abdominal aneurysm rupture	4 hours	Died in S-ICU
RH	63/M	Multiple total occlusions/ collaterals only/CABx5	1½ days	Alive and well
AB	46/F	Acute cardiogenic shock 3 weeks post-CABx2 and aneurysmectomy	5 days	Alive and fairly well
GR	50/M	Acute cardiogenic shock	15 minutes	Died in CCU
PE	49/M	Intermittent hypotension, unstable, high risk pre-MI, 99% pre-septal, 100% circ/CABx2	2 days	Alive and well

CASE REPORTS

Case 1: Acute Myocardial Infarction with Cardiogenic Shock. A 51-year-old male sustained an extensive anterior wall myocardial infarction (complicated by five days of hypotension) four months prior to admission to another hospital. He continued to be limited in his activities by severe dyspnea and angina despite intensive medical management. He was brought to our CCU with ECG changes of an acute inferior wall infarction. The patient was hypotensive with documented low cardiac output and high pulmonary wedge pressures. Recurrent ventricular fibrillation was a complicating feature. Balloon assist was instituted via the right groin under local anesthesia. With augmentation, the cardiac output dramatically increased, his sensorium cleared, urine output increased, and pulmonary artery wedge pressures fell to a normal range. Ventricular fibrillation did not recur. IABA augmentation allowed the additional use of nitroprusside with continued improvement of cardiac output. The balloon was removed four days later. Limited ambulation was possible after two weeks of slow recovery. The patient was able to leave the hospital three weeks post-infarction. He died suddenly at home four months later.

Case 2: Myocardial Preservation Post-Myocardial Infarction with Persistent Pain. A 50-year-old male had stable angina with little exercise limitation until three days prior to admission when he began to have chest pain at rest. He experienced severe anterior chest pain continuously for three hours and was admitted to the CCU. Morphine and isordil were administered without relief of chest pain. Blood pressure remained normal but with sinus tachycardia. Nitroprusside was started, but hypotension limited its use. IABA was begun. The chest pain disappeared, pulmonary congestion cleared, and urine output increased. Nitroprusside in moderate doses was used in conjunction with IABA. The patient remained stable without pain until he died suddenly twenty-seven hours following balloon insertion. Autopsy demonstrated a massive acute anterior-lateral myocardial infarction involving greater than 50 percent of the ventricular wall.

Case 3: Cardiac Surgery in the Presence of Critical High Risk Coronary Lesions. A 65-year-old man experienced exertional angina over a period of fifteen years with acceleration of symptoms over the past year. At the time of cardiac catheterization, he had nocturnal angina nightly and angina with minimal activity many times during each day. Treadmill testing, using the Bruce protocol,[6] brought on angina at one minute thirty seconds with associated extensive diffuse S-T depression. Angiogram demonstrated normal left ventricular contractility with severe three vessel disease: 95 per cent proximal occlusive lesion in the right (RCA), and total occlusions of the proximal left anterior descending (LAD) and both proximal obtuse marginal branches of the circumflex

coronary artery. All distal vessels filled from multiple collaterals. IABA was begun in the operating room under ketamine anesthesia and oxygen. Aorto-coronary saphenous vein bypass grafting (RCA, LAD, Diagonal, OM, OM_2—CABx5) was accomplished with good intra-operative graft flows. IABA was removed the second postoperative day; the patient was discharged from the hospital on the twelfth postoperative day. His result has been excellent; he is free of angina, on no medications, and has a normal functional capacity seven months after surgery.

Case 4: Cardiac Surgery in the Presence of Advanced High Risk Valvular Dysfunction. A 67-year-old male enjoyed excellent health most of his life although fifty years earlier his physician had told him that he had a heart murmur. There was no known history of rheumatic fever. Cardiac decompensation requiring digitalization was first apparent two years before surgery. Eight months prior to surgery, an acute inferior myocardial infarction occurred, which was complicated by a subsequent left hemiparesis thought to be on an embolic basis. The neurologic defects largely cleared, but an increase in heart size, progressive dyspnea, and profound fatigue ensued. At the time of his cardiac catheterization, the patient was confined to bed despite optimal medical management. Catheterization studies showed severe nonrheumatic mitral regurgitation with moderate pulmonary hypertension. Resting pulmonary wedge mean pressure was 24mmHg with "V" waves to 29mmHg. Left ventricular pressure was 80 /20; left ventricular contractility was only mildly impaired. The left atrium was massively enlarged. Additional coronary artery disease was also present with a 60 per cent narrowing of the left main coronary artery plus severe narrowing in the proximal RCA and LAD systems. A Swan-Ganz catheter was placed in the pulmonary artery the night prior to surgery. IABA was instituted in the operating room with ketamine and oxygen supplement. Mitral valve replacement (#31 Bjork-Shiley), tricuspid annuloplasty, and coronary artery bypass (RCA and LAD) were performed. Minimal doses of dopamine were discontinued a few hours after surgery. The balloon was discontinued the fourth post-operative day. Recovery was slow but dramatic. The long-term result has been excellent; the patient is walking two miles a day without fatigue six months after surgery.

Case 5: Failure to Wean Off Cardiopulmonary Bypass. A 47-year-old female had experienced angina for five years with progressive limitation despite maximal doses of coronary dilators and propranalol. Cardiac catheterization showed normal ventricular function with significant proximal lesions in the obtuse marginalis and posterior descending coronary arteries. Double saphenous vein bypass was performed with good graft flows. Fifteen minutes after bypass had been discontinued, the patient became hypotensive. Bypass was reinstituted. The patient could not be weaned from cardiopulmonary support despite a period of myocardial "rest", multiple pharmacologic interventions, and reconstruction of the saphenous vein-posterior descending anastomosis. IABA was instituted via the right groin and continued until the fifth post-operative day. Electrocardiographic findings of nontransmural inferior wall injury evolved. Marginal cardiac output gradually improved, and the patient was discharged from the hospital on the eighteenth postoperative day. Six months later she enjoys near normal functional capacity.

Discussion

The benefits of intra-aortic balloon assist have been amply documented in various publications.[4, 7] It has proven to be an excellent modality for reversing cardiogenic shock, supporting a failing myocardium after cardiopulmonary bypass, and increasing patient survival in high risk cardiac surgery. During diastole, the rapid displacement of blood from the aortic arch during balloon inflation results in an increased diastolic pressure and increased coronary and systemic blood flow. Improvement in cardiac output, cerebral blood flow, and renal function are usually seen. In systole, the balloon deflates rapidly and decreases ventricular afterload, thereby decreasing the work load and oxygen demand of the myocardium. Thus, the heart muscle enjoys the delivery of more oxygen and substrate for less work. Consequently, there is a reduction in angina. Simultaneously, there is a decrease in left ventricular filling pressure and pulmonary wedge pressure with reduction in pulmonary congestion and dyspnea. The thirty percent reduction of left ventricular energy consumption results in the opportunity for the overstressed heart to recover,[4] and further infarction may be limited. With early implementation of IABA, the correctable causes of left ventricular failure may be reversed. The dramatic but temporary benefits of IABA may allow the means and the time for myocardial recovery.

Buckley first used IABA in a series of twenty-six patients who were unable to come off cardiopulmonary bypass after all other modes of support had failed. From this group of patients, in which the expected mortality was nearly 100 percent,

there were ten long-term survivors.[8] This remarkable salvage encouraged others to widen the indications for IABA. Initial enthusiasm for the use of IABA in cardiogenic shock following acute myocardial infarction has been dampened because long-term salvage rates have been generally poor.[9] Our experience supports this conclusion. An increasingly aggressive approach toward patients in deep cardiogenic shock secondary to an acute myocardial infarct has recently been formulated. While IABA support is continued, diagnostic angiography is performed and appropriate bypass surgery follows. Even among the highest risk patients in whom IABA cannot be terminated after forty-eight hours, survivors have been reported. One series describes six long-term survivors from a group of thirteen IABA-dependent patients in whom emergency bypass surgery followed angiographic demonstration of potentially correctable coronary lesions.[10] There is ample evidence to suggest that an aggressive approach to desperately ill patients in cardiogenic shock in a community center can be successful.

While the need for dramatic intervention is most apparent in cardiogenic shock, an additional role for IABA exists in early prophylactic use for perioperative preservation of myocardium. More frequent elective IABA use in the operating room is anticipated. This use will protect jeopardized myocardium during the so-called "critical period" of anesthesia induction[11] before the establishment of improved cardiac blood flow. Recent reports have demonstrated dramatic reductions in morbidity and mortality in this high risk group of surgical candidates.[12 - 15] We foresee increasing IABA use in patients with acute myocardial infarction whose cardiac pump function remains adequate but whose continuing pain and enzyme changes suggest widening areas of ischemia.

Although the device has been employed in less than 10 percent of our surgical cases, we feel IABA a necessity for a complete regional cardiac care unit. Appropriately applied intra-aortic balloon assistance allows for an increased and long-lasting salvage of the appropriately selected, desperately ill cardiac patient.

ACKNOWLEDGEMENT

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Pulmonary embolism with embolectomy

REPORT OF A CASE

Herman D. Sorensen MD, Billings, Montana

Pulmonary embolism is common, difficult to diagnose, and often difficult to manage. It has been estimated that there are 142,000 episodes of fatal pulmonary emboli and 285,000 cases of non-fatal pulmonary emboli in the United States yearly. The rate of hemorrhagic complications of anticoagulant treatment was 27 per cent in the Urokinase Pulmonary Embolism Study, and has been reported elsewhere at two to 15 per cent. Because of this high incidence of complications, care should be taken to avoid misdiagnosis. Underdiagnosis, however, must also be avoided. Pulmonary arteriography is the gold standard for diagnosis, but is not always available, and itself carries a small risk.

To illustrate some of the problems in diagnosis and management of this disease, report of a recent case is presented.

CASE REPORT

A 17-year-old girl who had been taking oral contraceptives for acne, developed lower abdominal pain. She was hospitalized and treated with intravenous antibiotics. The pain continued, but was much less severe. She was sent home. A week following onset of the pain, while sitting on the couch at home, she became suddenly dyspnic, acutely ill, and began to clutch her chest. She was taken to her local hospital where diagnosis of pulmonary embolism was suspected. She was given 10,000 units of Heparin IV and transferred to Billings.

Physical examination on admission showed an acutely ill, dyspneic, cyanotic girl. Blood pressure was 100 /50. Pulse rate was 164. The pulse was thready, and the jugular veins were prominent. The chest was clear. The heart sounds were partially obscured by heavy breathing but P-2 seemed prominent. The liver was palpable 4 to 5 cm below the right costal margin, and was quite tender.

Blood gases on admission were pH 7.37, pO_2 43, pCO_2 42, HCO3 23. Electrocardiogram showed right axis deviation, and nonspecific ST-T abnormalities. Chest x-ray showed a difference in vascularity between the right and left lung. Lung scan showed pulmonary perfusion to be limited to the right upper lobe with no significant perfusion present in the left lung.

The patient was treated with high flow oxygen, and continued on heparin. Over the next several hours she continued to deteriorate. She became hypotensive, and eventually, even on dopamine, had no audible blood pressure. Urine output dropped to zero. Blood values were increasingly abnormal to the point of pH 7.17, pO_2 61, pCO_2 35 and HCO −11, consistent with hypoxemia and severe metabolic acidosis.

The patient was taken to the operating room and put on cardiopulmonary bypass. Because of confidence in the diagnosis of massive pulmonary embolism pulmonary angiography was not performed. The pulmonary artery was opened, and several very large well formed clots were removed. Blood gases drawn immediately on return to the ICU were pO_2 211, pCO_2 27, pH 7.41, and HCO3 −16. Her subsequent course was complicated by acute tubular necrosis and infarction in the left lower lobe with prolonged fever, but she eventually achieved full recovery, and was discharged on coumadin.

Discussion

The diagnosis in this case was straightforward. The patient was previously healthy but had a pre-disposing condition, namely she was on oral contraceptives. She became suddenly acutely ill, had typical physical findings of right heart failure, typical ECG changes of right heart strain, and characteristic x-ray and lung scan findings of pulmonary embolism. However, the diagnosis is not always easy. Most patients suspected of having pulmonary emboli have underlying disease, particularly cardiac, and they are often elderly. These characteristics

can confuse the physical findings as well as the ancillary tests, and thus make diagnosis difficult.

Diagnostic aids, in addition to history and physical examination, to evaluate the problem are blood gases, chest x-ray, lung scan, and pulmonary arteriogram. The electrocardiogram is also helpful in certain circumstances. Szucs and his associates in a prospective study of fifty patients with angiographically proven pulmonary emboli found that the pO_2 was less than 80 mm Hg in all patients and none had normal lung perfusion scan. Their conclusion was that a normal lung scan or pO_2 greater than 80 on room air essentially ruled out pulmonary emboli. In the Urokinase Pulmonary Embolism Trial, however, the pO_2 was greater than 80 in twelve per cent of patients not receiving O_2, and some of these patients had sizable pulmonary emboli. The lung scan was abnormal, however, in all patients in the Urokinase Trial who had lung scans.

Other studies corroborate that the lung scan is a sensitive screening test for pulmonary emboli. Specificity, however, is a problem. The specificity can be improved by several methods. Moses and others reported 104 cases of pulmonary embolism with scans and angiography. They interpreted the scans as highly probable when segmental or lobar perfusion defects presented. Thirty-seven of 45 patients with angiographically proven pulmonary emboli were found to have highly probable scans, and 50 of 59 patients without emboli had low probability scans. When scan findings were correlated with x-ray findings, percentages improved. Suggestive x-ray findings included infiltrate effusion atelectasis, and elevated hemi-diaphragm. Of fifteen patients with low probability scans and no x-ray evidence of pulmonary emboli none had emboli, and of 43 patients with highly probable scans and suspicious x-rays, 38 had emboli. If x-ray and scan pointed in the same direction it was concluded a correct diagnosis could be made.

Another method of improving the specificity of the lung perfusion scan is to perform a simultaneous ventilation scan. This is done by having the patient breathe radioactive xenon gas, and measure the washout of the gas by a scintillation camera. Poorly ventilated areas have slow washout. If a ventilation defect exists in the area of perfusion defect, pulmonary embolus is less likely. Likewise, pulmonary emboli are more likely if a perfusion defect exists in the absence of a ventilation defect.

Any doubts about diagnosis should be resolved by pulmonary angiography. This is the definitive test. It is relatively safe. No fatalitites were found in 310 pulmonary angiograms done in the Urokinase Trial. One cardiac perforation and five cases of ventricular tachycardia required treatment. Other reports substantiate this low complication rate. Deaths, however, have been reported and are particularly likely with massive emboli and with severe pulmonary hypertension. Pulmonary angiography should not be performed in the presence of pulmonary hypertension. Therefore, right heart pressures should be measured prior to pulmonary angiography.

Heparin is the mainstay of treatment for pulmonary emboli. Use of very high doses of heparin in massive pulmonary emboli is advocated, as evidence exists that in these doses, in addition to the anticoagulation effect, bronchoconstriction, and pulmonary vascular resistance are reduced. All patients in whom there is no contraindication should be heparinized as initial treatment. The Urokinase Trial showed certain superiorities of Urokinase to heparin. Significant but slight differences were noted. These were limited to early resolution of the emboli as determined by serial angiogram and lung scans. No difference in mortality was seen in the two groups. The hemorrhagic complication rate was somewhat higher in the Urokinase group. Urokinase is not currently available for general use, making heparin initial treatment of choice.

Inferior venacaval ligation has been advocated in certain cases of pulmonary emboli. Common complications from this procedure include chronic lower extremity venous stasis, fluid redistribution with

lowered right heart pressures, decreased cardiac output, oliguria, and hypotension. Mortality rates in the chronically ill, even with the use of the umbrella filter, remain fairly high. This procedure should be limited to patients who have documented pulmonary emboli on adequate anticoagulation therapy, or who have contraindication to anticoagulation treatment.

Pulmonary embolectomy is a controversial procedure. Mortality remains high, ranging from 35 to fifty per cent. In general the indication is hypotension unresponsive to vasopressors in a patient deteriorating on heparin treatment. This is a select group of patients. Most patients can be supported temporarily until clot lysis improves the situation. In the Urokinase Trial mortality rate for pulmonary embolectomy with associated shock was 36 per cent, and this rate might lead one to conclude that pulmonary embolectomy is never indicated. However, as part of the study protocol, physicians were discouraged from admitting patients in whom pulmonary embolectomy was being considered. The more seriously ill patients potentially salvageable by embolectomy were, therefore, at least partially screened out of the study. Pulmonary embolectomy remains a potentially life saving procedure, as demonstrated in the case presented here, though indications are rare.

Summary

The diagnosis of pulmonary embolism is difficult but can usually be made with good accuracy utilizing chest x-ray, lung scan and clinical considerations. Pulmonary angiography is often indicated and is safe. Heparinization is the treatment of choice but where heparin is ineffective or contraindicated, IVC ligation should be considered. In massive pulmonary embolism with shock, unresponsive to heparin, pulmonary embolectomy may be lifesaving.

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Preinfarction angina: evaluation and treatment

John R. Burg, MD, Billings, Montana

This clinical syndrome may portend myocardial infarction or sudden death; aggressive medical and surgical management is warranted.

Coronary atherosclerosis presents a clinical spectrum ranging from angina pectoris to myocardial infarction. An ill-defined syndrome is also recognized which is intermediate in severity between these two manifestations. This entity[1] has been variously referred to as the preinfarction syndrome, impending myocardial infarction, unstable angina pectoris, status anginosis, acute coronary insufficiency and the intermediate coronary syndrome. The diverse terminology used to describe this clinical picture suggests that none is acceptable to all physicians. However, because risk of subsequent myocardial infarction or death is higher in this syndrome[2] than in chronic stable angina pectoris,[3] the term "preinfarction angina" will be used in this report to denote this clinical state, even though it is clear that infarction occurs in only a minority of these patients.[2,4,5] It is of interest that this syndrome accounts for almost twenty per cent of admissions to coronary care units.[4]

Preinfarction angina must be considered a medical or surgical emergency.[6] Characteristic is a severe cardiac ischemic episode lasting fifteen or more minutes. The onset is usually unrelated to exertion or other known precipitating factors. Its appearance must be considered a major change in the clinical status of the patient. Although discomfort may appear suddenly in a previously asymptomatic subject, more frequently there is an accelerated symptomatic pattern in a patient known previously to have stable angina pectoris. The episodes may be more frequent or severe, or they may occur with less provocation or at rest.[7] Tachycardia and hypertension are frequently observed during periods of discomfort.[8] Moreover, transient electrocardiographic (ECG) changes such as ST segment depression or elevation with T wave inversion are not unusual. Arrhythmias may be documented. Cardiac enzymes remain normal or at least rise no more than fifty per cent above the normal range.[5] New Q waves or loss of precordial R waves are incompatible with the diagnosis of this syndrome and indicate transmural myocardial infarction.[4]

A major obstacle confounding the analysis of the preinfarction syndrome has been the use of nonuniform criteria in diagnosis. Assessment of the natural history of this disorder is difficult since information is scarce and unreliable. The true natural history will never be known since all affected recognized individuals receive some form of medical management. Comparison of one series to another is not often valid. For example, the risk for patients with ECG changes is higher than those with stable electrocardiograms;[2] some studies[4,5] require ECG changes during symptoms for inclusion and are not comparable to those which accept a less rigorous definition. To date, results of two incomparable prospective trials have been published,[5,9] and another is awaited with anticipation.[10]

Medical therapy has revolved about hospitalization with bed rest and treatment with oxygen, nitrites and beta-blocking agents such as propranolol. Anticoagulants are no longer widely employed, their efficacy never having been conclusively substantiated. With medical treatment, symptoms can be controlled acutely in approximately eighty-five per cent of patients hospitalized.[4] Myocardial infarction develops acutely in about seven per cent of patients so treated,[2] but the long-term outlook is more ominous and is similar to or worse than that of transmural myocardial infarction.[1,2] Thus, the development of late infarction has been reported to occur in sixteen to fifty per cent of patients having had symptoms consistent with preinfarction angina.[11] The associated mortality is said to be thirty per cent.[1]

Because of the life-threatening nature of the preinfarction syndrome, surgery is frequently considered in the aggressive management of these patients. In most major medical centers, surgical therapy currently is the preferred mode of treatment.[6] Surgical interventions encompass the use of the intra-ortic balloon pump and coronary artery bypass surgery. The intra-aortic balloon pump is highly effective in relieving ischemic symptoms and stabilizing the most critical patients prior to coronary arteriography and bypass surgery.[12] Symptoms in most patients, however, will be relieved utilizing medical therapy and the intra-aortic balloon pump need be used only rarely.

In the past, attention has been called to an increased morbidity or mortality from coronary arteriography.[7] This increased risk is not universal, and in the hands of the experienced angiographer, the study can be completed safely.[9,13] Arteries containing a seventy per cent or greater stenosis are considered significantly diseased. Double and triple vessel disease is more common than single vessel disease.[10] Ten per cent of patients with preinfarction angina have no significant demonstrable disease. Collateral circulation to the affected arteries is less common than in patients with chronic stable angina.[4]

Coronary artery bypass surgery has an immediate appeal in that it is the only known form of therapy which increases blood flow to the ischemic myocardium. Most centers continue to utilize saphenous vein grafts. The operative mortality for bypass surgery is variable but in most reported series averages five to ten per cent.[4,5,13,14] This figure is slightly higher than that for chronic stable angina pectoris. Intraoperative myocardial infarctions occur in eleven to fifteen per cent of patients.[5,13,15] Nonetheless, the long-term results following surgery have been most gratifying. The probability of survival at the end of three years in one surgically treated series[15] was ninety-three per cent. Most patients attain functional Class I or II (New York Heart Association) and have objective improvement in exercise tolerance and ECG abnormalities as demonstrated by exercise testing or atrial pacing.[5] In contrast, medically treated patients have not only a higher subsequent mortality rate but are also more likely to have disabling angina pectoris with impaired functional classification. Most patients are able to return to work postoperatively. Graft patency at six months averages seventy-five to eighty-five per cent, figures not differing significantly from those obtained in patients with stable angina pectoris.[13,15] With the exception of one study,[16] ventricular function is not improved significantly.[7,15]

The Billings Experience

Coronary artery bypass surgery has been performed in Billings, Montana, since July, 1972. The case load has increased dramatically during the last two years. Patients with preinfarction angina comprise one-third of the series. Early in our experience, we performed coronary arteriography urgently and patients were transported directly to the operating suite. Because of four operative deaths occurring over a relatively short period of time, we reassessed our management of this syndrome. Since early 1974, we have

attempted to stabilize the patient with propranolol and nitrites, performing coronary arteriography and surgery on an elective basis. Those few patients who continue having pain undergo coronary arteriography and urgent surgery.

Since adopting this policy, our operative mortality has diminished significantly. No operative deaths have occurred among the subsequent 140 men operated for either stable or preinfarction angina. In keeping with another series,[17] the operative mortality for women is higher. Other centers have also stressed the rationale of this management program.[4,5]

Summary

The preinfarction syndrome demands prompt medical or surgical intervention. With medical management, long-term morbidity and mortality are significant. Coronary artery surgery is feasible in many of these patients with an acceptably low mortality rate and offers the prospects of prolonging life, relieving ischemia, and improving the patient's quality of life.

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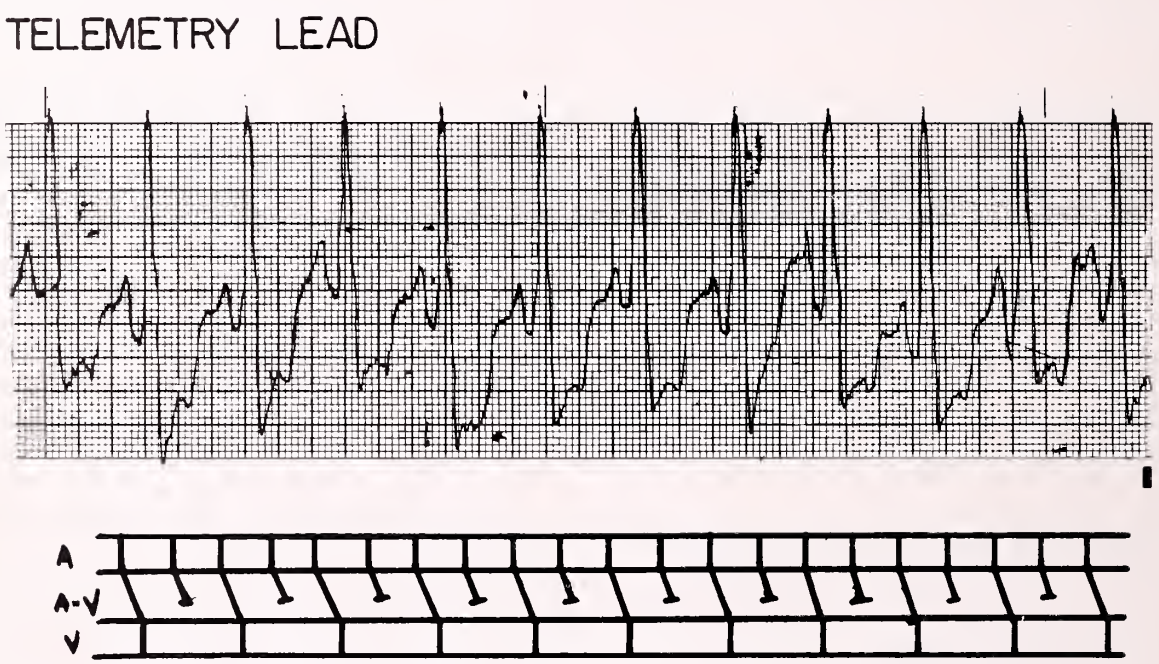
what's your reading?

From Page 83



Answer:

A brief initial analysis reveals a tachycardia (104 beats /min.) with P-waves preceding an abnormally wide QRS. The first guess would be sinus tachycardia in a patient with an underlying intraventricular conduction delay and marked ST depressions. However, careful scrutiny of the ST-T wave suggests a P-wave deforming the upsloping ST segment. These march out at exactly twice the more obvious P-P interval, and suggest that this is an atrial or supraventricular tachycardia with a rate of 208 and 2:1 A-V block, resulting in a ventricular response of half the atrial rate. Although the telemetry recording is technically poor, distortion of the ST segment is seen consistently, most likely representing the almost obscured P-waves. This ryhthm soon degenerated into a profound sinus slowing with idioventricular beats (not shown).

Bone grafting in comminuted hip fractures*

D.W. Grimes MD and R.W. Garner, MD, Boise, Idaho

During the years of the twentieth century, great strides have been made in treatment of fractures of the hip. At the turn of the century amputation was still in vogue in many areas of the world, or "skillful neglect" was utilized. Later, traction devices and casting were introduced, to be followed by open reduction and internal fixation. The advent of antibiotics changed the whole rationale of treatment of fracture. Phraseology such as "making a cesspool out of a closed fracture," "never add bone graft until Mother Nature has been given a chance," and similar out-moded cliches are, in our opinion, no longer valid.

For an appreciable period of time the authors have independently utilized methods as suggested by Hughston[1] and Sarmiento[2] and others in the fixation of comminuted intertrochanteric fractures of the hip. The success rate has been good and, although it is not being presented, we believe it compares favorably with the above authors.

Fig. 1: Preoperative X-ray of an elderly man with osteopenic bone.

CASE HISTORY

A 72-year-old man was admitted to the hospital on December 5, 1975, after a period of three days at another facility. He had sustained an intertrochanteric fracture of the hip (Fig. 1). At the time of arrival he was disoriented and suffered from an intestinal ileus. After five days of nasogastric decompression, fluid therapy, and antiobiotics, a Jewett nail was inserted into the hip with the patient in the lateral decubitus position. The preoperative prophylactic cephalosporin was augmented at surgery by the addition of the antiobiotic intravenously, and the two were used for one week postoperatively. After the completion of the nail fixation, ⅛ inch strips of iliac crest bone were removed by a separate incision. The time taken to accomplish this was less than fifteen minutes. The graft material was inserted anteriorly and posteriorly in a palisade manner at the fracture site. A hemovac was put in place.

The following day the patient was sitting in a bedside wheelchair. Prophylaxis against thrombophlebitis was commenced on the day of surgery by leg massage and passive joint motion performed by

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the ward personnel. Good healing by X-ray was manifest during the fourth week. The tilt table was started twice daily with progression to ambulation in parallel bars and thence to assistive aids by the physiotherapy department. Postoperative films taken during the sixth week (Fig. 2) show good bone deposition. The patient was released on Canadian crutches, walking unassisted. No limitation of motion, weakness or other undesirable conditions were noted.

The above case report is considered typical of the results of the authors by means of the simple addition of strips of iliac bone around the fracture site. No infections have been encountered with the precautionary antibiotic regimens utilized.

A plea is made for second thoughts concerning the routine addition of bone graft to comminuted intertrochanteric and subtrochanteric fractures of the hip. This simple and safe procedure should be the rule rather than the exception. Under the antiobiotic umbrella infections have been non-existent. Early weight bearing is readily accomplished from the stimulus given by the grafting.

Fig. 2: The same patient six weeks after surgery. The patient was up and walking with a cane.

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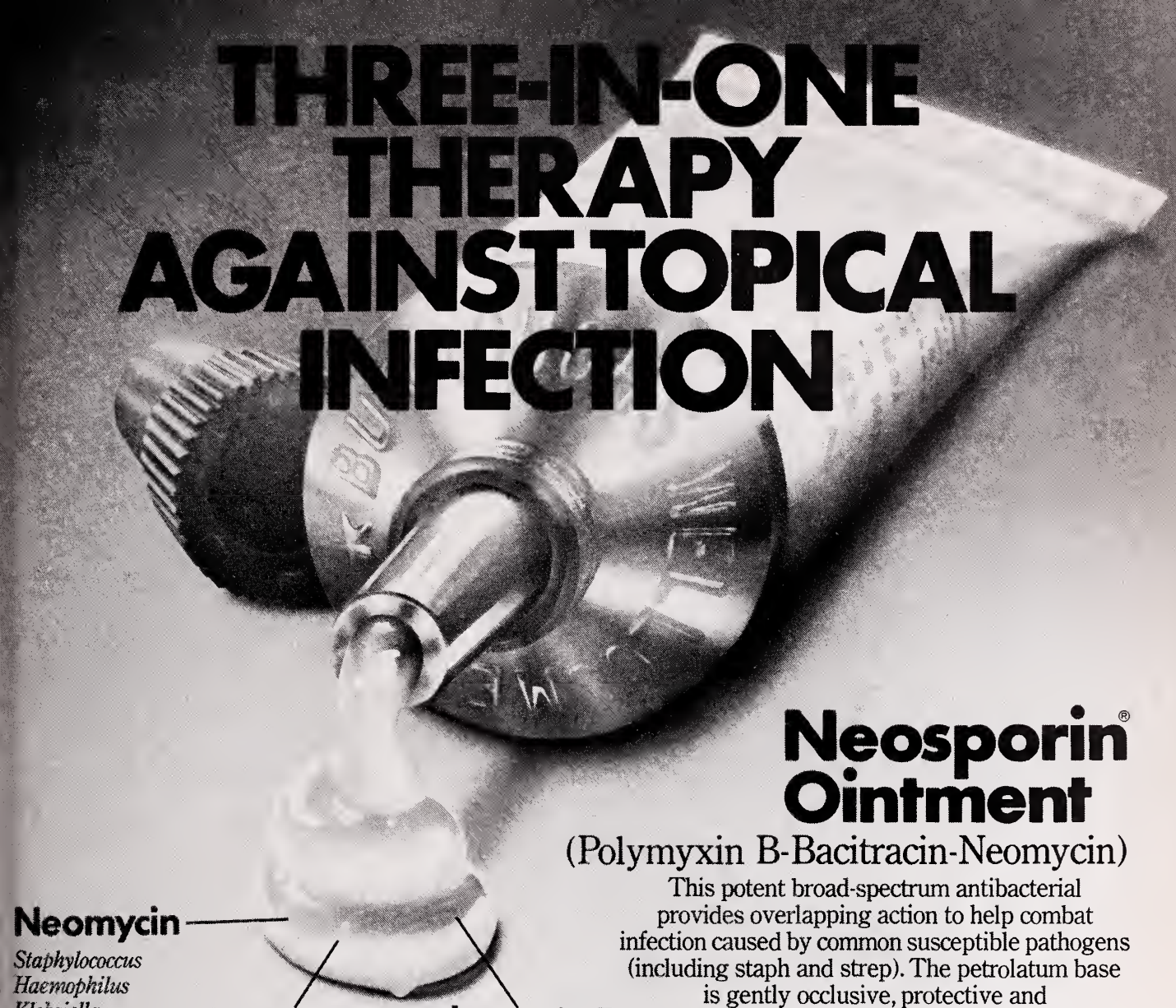
Arch over the Railroad, Bordentown.

THREE-IN-ONE THERAPY AGAINST TOPICAL INFECTION



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When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

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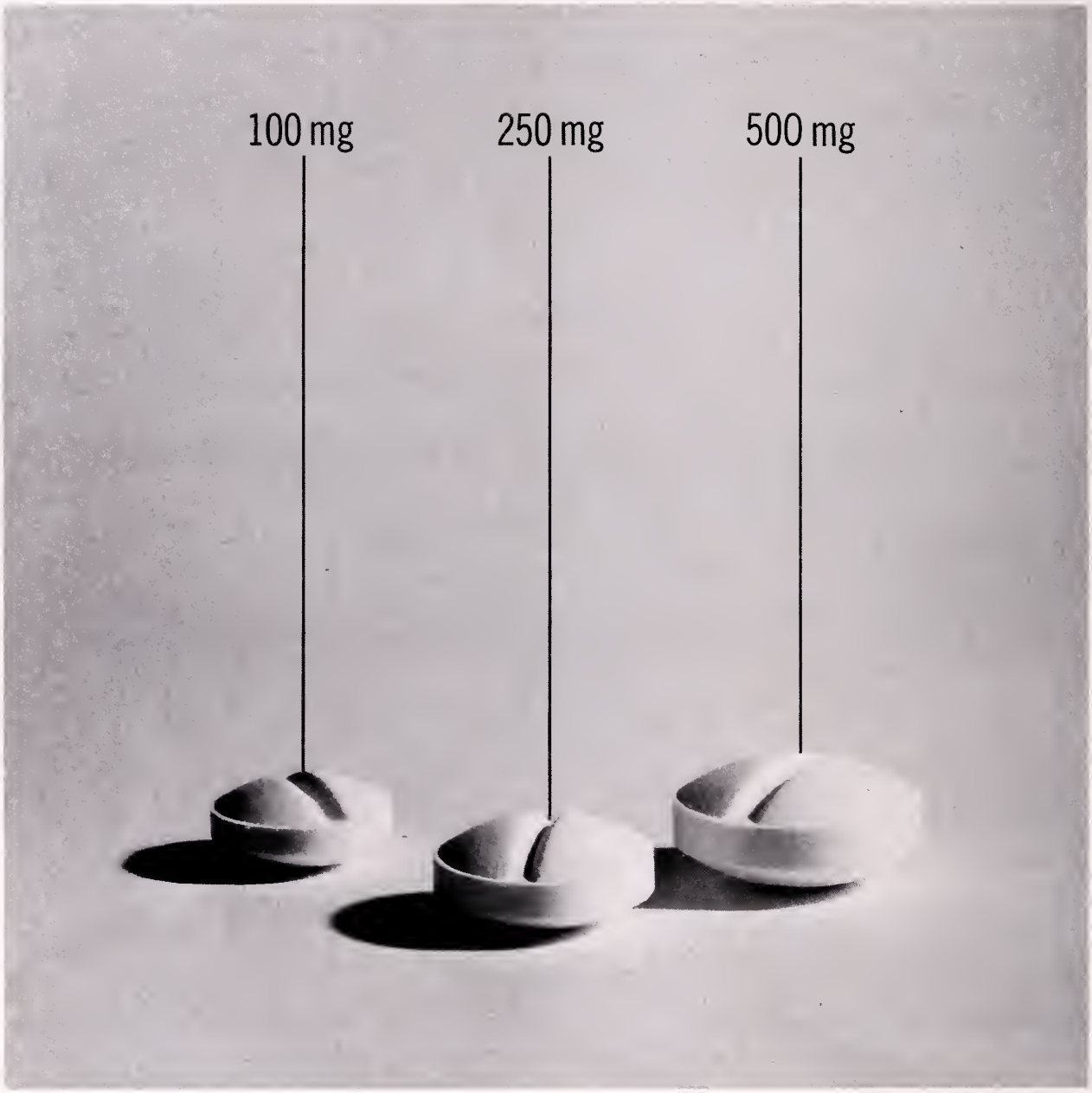

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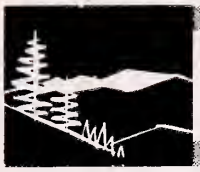

COLORADO

Coloradans Named to Chest Physicians

250 physicians and surgeons were recently inducted into the American College of Chest Physicians on the occasion of the 42nd Annual Scientific Assembly of the organization. Among those from Colorado who were inducted were Sheldon L. Spector, MD, Denver, and Milton L. Wiggins, MD, Colorado Springs.

Colorado Doctors Elected to AAOS

Among those inducted as Fellows of the American Academy of Orthopaedic Surgeons on February 4, during the 44th Annual Meeting of the AAOS in Las Vegas, Nevada, were Clifford J. Rome, MD, Greeley, David P. Fisher, MD, Grand Junction, and Lawrence G. Gigliotti, MD and John M. Provost, MD, both of Colorado Springs.

MEDICAL SCHOOL NOTES

University of Colorado School of Medicine

Oxford Lecture Ahead for Naughton

Michael A. Naughton, PhD, professor in the Division of Perinatal Medicine at the University of Colorado School of Medicine, has been invited to spend a week at Oxford University in England as a resident Litchfield Lecturer, and he will spend time with the Nuffield Department of Clinical Medicine. The Third Earl of Litchfield established the trust in the reign of George III, under which a sum of money was bequeathed to the University on the condition that income should be used for clinical instruction in Oxford for members of the University.

Cummings and Claman Awarded Sabbatical Purses

Donald J. Cummings, PhD, professor of microbiology, and Henry N. Claman, MD professor of microbiology and medicine, both at the University of Colorado School of Medicine, have received Macy Foundation Faculty Scholar Awards of $25,000 each to finance one-year sabbaticals to follow special studies.

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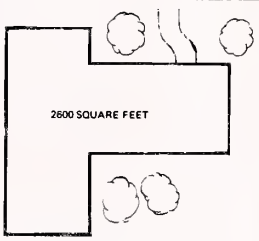
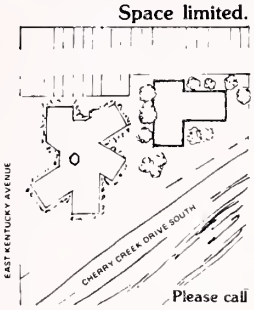


Completion planned
for summer
1977.

Space limited.



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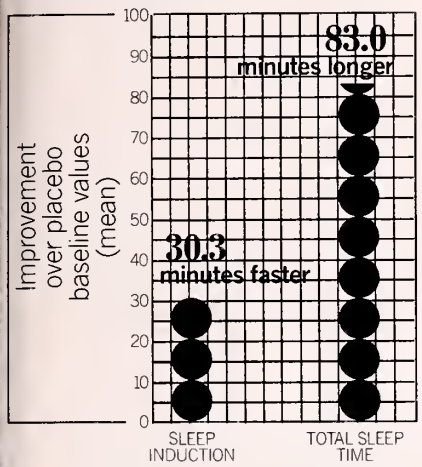
Six female insomniacs, ranging in age from 67 to 82 years received Dalmane (flurazepam HCl) for seven consecutive nights in the sleep research laboratory.[1] Improvement over pre-treatment baseline levels was significant for sleep induction and sleep maintenance ($p < .05$). And the greater the sleep problem in these patients, the better the effect with Dalmane (significant correlation at $p < .01$ level)

Elderly insomniacs fell asleep faster, slept longer[1]



Results expand and confirm objective proof of efficacy in younger adults with insomnia

The effectiveness of Dalmane (flurazepam HCl) was demonstrated in earlier studies of 32 younger adults with trouble falling asleep, staying asleep or sleeping long enough.[2] On average, in these studies, Dalmane induced sleep within 17 minutes and provided 7 to 8 hours of sleep, at the same time reducing number of nighttime awakenings.

Relative safety, even in patients on warfarin

Morning "hang-over" has been relatively infrequent with Dalmane. And no unacceptable fluctuation in prothrombin time has been reported in warfarin patients on Dalmane.[2,3] The usual adult dosage is 30 mg *h.s.*; in elderly and debilitated patients, limit initial dosage to 15 mg to help preclude oversedation, dizziness or ataxia.

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: **Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

REFERENCES:
1. Frost JD Jr: Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
2. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
3. Robinson DS, Amidon EL: Interaction of benzodiazepines with warfarin in man, in *The Benzodiazepines*, edited by Garattini S, Mussini E, Randall LO. New York, Raven Press, 1973, p. 641

Objective evidence proves insomnia relief in elderly patients

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THE BOOK CORNER

New books received are acknowledged in this section and such acknowledgment must be regarded as sufficient return for the courtesy of the sender. Selection will be made for review in the interests of our readers and as space permits. Books are listed with advance data supplied by publishers. Prices quoted are not guaranteed. For further information, address queries to the publishers. Books here listed are available for lending from the Denver Medical Society Library.

Recent Acquisitions

The Abusing Family: Blair Justice. New York, Human Sciences Press, 1976. 288 p. $14.95.

Acute Diarrhoea in Childhood: Symposium on Diarrhoea in Childhood, London, 1975. 375 p. (Ciba Foundation Symposium, new series; 42) Gift.

Battered Wives: Del Martin. San Francisco, Ca., Glide Publications, 1976. 269 p. $6.95.

Beck's Obstetrical Practice and Fetal Medicine: E. Stewart Taylor. 10th ed. Baltimore, Williams & Wilkins, 1976. 639 p. $38.50.

Cancer of the Skin: Rafael Andrade, et al. Philadelphia, Saunders, 1976. 2 v. $90.00.

Cardiac Diagnosis and Treatment: Noble O. Fowler. 2nd ed. New York, Harper & Row, 1976. 1150 p. $42.50.

Colposcopy: Malcolm Coppleson, Ellis Pixley and Bevan Reid. 3rd printing. Springfield, Ill., Thomas, 1976. 346 p. $21.50.

Controversy In Surgery: Richard Varco and J.P. Delaney, ed. Philadelphia, Saunders, 1976. 713 p. $29.00.

Coronary Artery Disease: Richard Gorlin. Philadelphia, Saunders, 1976. 317 p. (Major problems in internal medicine, v. 11) $16.50.

Current Obstetric and Gynecologic Diagnosis and Treatment: Ralph C. Benson. Los Altos, Cal., Lange, 1976. 911 p. $16.00.

Forward Plan for Health-FY 1978-82: U.S. Department of Health, Education and Welfare. Public Health Service. Washington, D.C., August, 1976. 137 p. $1.90.

Fractures and Joint Injuries: J.N. Wilson, ed. 5th ed. New York., Churchill-Livingstone, 1976. 2 v. $95.00.

The Great American Medicine Show: Spencer Klaw. New York, Viking Press, 1976. $11.95.

Harrison's Principles of Internal Medicine; Tinsley R. Harrison, ed. 8th ed. New York., McGraw-Hill, 1977. 2088 p. $37.00.

Head Injuries: Robert L. McLaurin, ed. New York, Grune, 1976. 301 p. $12.50.

Health Care in a Changing Setting: Symposium on U.K. Health Needs in a Changing Setting, Ciba Foundation, 1975. New York, Elsevier, 1976. 188 p. Gift.

The Impaired Physician; American Medical Association. Chicago, AMA, 1975. 54 p. Gift.

Management of the Unconscious Patient: William R. Darmody. St. Louis, Mosby, 1976. 120 p.

The library has begun a monthly awareness service, "Keeping Current", in the socio-economic area. Topics covered include cost of health care, evaluation and quality of health care, and consumer participation. Contact the library at 534-8580 if you wish to be placed on the mailing list.

Manual on Control of Infection in Surgical Patients: Americal College of Surgeons. Committee on Control of Surgical Infections. Philadelphia, Lippincott, 1976. 280 p. $16.00.

Now That You've Incorporated: Sheldon H. Gorlick. Oradell, N.J., Medical Economics, 1975. 176 p. $10.00.

Of Love and Lust: Theodor Reik. N.Y., Aronson, 1974. 623 p. $15.00.

Operative Surgery: Nose and Throat: John Ballantyne, ed. 3rd ed. Boston, Butterworth, 1976. 389 p. $57.00.

Ophthalmic Plastic Surgery: Sidney A. Fox. 5th ed. N.Y., Grune & Stratton, 1976. 666 p. $29.50.

Patient Flow Patterns and Hospital Service Areas in Colorado: Colorado/Wyoming Regional Medical Program. Denver, RMP, 1976. 197 p. Gift.

Pediatric Clinical Gastroenterology: Claude Roy. 2nd ed. St. Louis, Mosby, 1975. 799 p.

Physical Examination of the Spine and Extremeties: Stanley Hoppenfeld, N.Y., Appleton-Century-Crofts, 1976. 276 p. $16.50.

Physician Resources in Colorado: Colorado/Wyoming Regional Medical Program. Denver, RMP, 1976. 256 p. Gift.

Pituitary and Parapituitary Tumors: John Hankinson, et al. Philadelphia, Saunders, 1976. 217 p. (Major problems in neurology, v. 6)

Post-Physician Era: Jerrold Maxman. N.Y., Wiley, 1976. 300 p. $15.95.

Rehabilitation Medicine: P.J.R. Nichols. Boston, Buttersorths, 1976. 311 p. $16.95.

Research and Medical Practice: Symposium on Research and Medical Practice, London, 1976. N.Y., Elsevier, 1976. 220 p. (Ciba Foundation Symposium, new series; 44) Gift.

Stress in Health and Disease: Hans Selye. Boston, Butterworths, 1976. 1256 p. $79.50.

Trends Affecting the U.S. Health Care System: U.S. Department of Health, Education and Welfare. Washington, D.C., U.S. Government, 1976. 430 p.

Tumors of the Eye: Algernon B. Reese. 3rd ed. N.Y., Harper & Row, 1976. 477 p. $50.00.

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OBITUARIES

Colorado

Henry Hall Zeigel, MD
98th Past President
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Former president of the Colorado Medical Society (1967-68), Doctor **Henry Hall Zeigel** died December 18, 1976. He was 77, having been born September 30, 1899 in Marshall, Missouri. His undergraduate education was received from the University of Washington, and his MD from the Kansas City College of Medicine and Surgery. He received his license in 1924. After an internship at the Latter Day Saints Hospital at Salt Lake City he settled at Collbran, Colorado to practice, and remained there until his death, forty-nine years later.

He was a member of the Mesa County Medical Society (past president) and of the AMA. Doctor Zeigel was active in medical society affairs, having served on as well as chaired, several important committees for both the Mesa County and the state societies.

He was a member of the Selective Service Board during World War II, and was active in his own hospital, the Plateau Valley Congregational Hospital at Collbran, and was affiliated with the Meeker Pioneer Hospital, Rifle Community Hospital, and St. Mary's Hospital, Grand Junction. He was a pioneer member of the American Academy of General Practice.

He was married to Margaret Amelia Stoufer in Liberal, Kansas, July 12, 1930. She survives the Doctor as do a daughter, Mrs. Joyce Marie Bean of Richland, Washington, a son, Dr. John Stoufer Zeigel of Grand Junction, and three grandchildren.

* * *

Doctor **Zdenka Kalendovsky,** Denver, was slain by an unknown assailant, Monday, February 7, 1977, as she left her office at The Denver Clinic to go to her car.

Doctor Kalendovsky was a Czechoslovakian who fled that country with her husband in the early 1960's, coming to the United States. About four years ago, after having completed a neurological fellowship at Cornell University in New York, she came to the University of Colorado Medical Center for more graduate training.

At the time of her death she was an Assistant Clinical Professor at the University of Colorado, and was on the staffs of both the Veterans Administration and Colorado General hospitals. About two years ago she became associated with the Denver Clinic at 701 East Colfax Avenue in Denver.

Doctor Kalendovsky had made substantial contributions in neurological research. She had been awarded the Harold Wolf Prize by the American Association for research in Headache. Her most significant work had to do with those neurological conditions related to the cerebral vascular system, such as strokes and vascular headaches. She was particularly concerned with those neurological complications thought to be associated with contraceptive pills.

Doctor Kalendovsky was born in Brno, Czechoslovakia, February 15, 1936, and was educated in that country. She was formerly the Czech women's national tennis champion. She and her engineer husband, Jiri, lived in Boulder, Colorado with their daughter.

* * *

Joseph H. Bechtold, MD, died Tuesday, December 28, 1976, in Denver after a short illness.

He was born April 20, 1915, in Denver, and attended Regis High School and College, following which he attended the University of Colorado Medical School where he received his MD.

After an internship served at Tacoma General Hospital, Tacoma, Washington, Doctor Bechtold opened an office for the general practice of medicine in Denver.

He was a member of the staff at Mercy Hospital, and was one of the doctors for Union Pacific Railroad. He was a member of the Denver and Colorado medical societies and of the AMA.

He was married to Eva M. Laursen in Denver on June 25, 1944. She survives him as do five daughters, Mrs. Karen Gifford and the misses Ann, Peggy, Kay and Amy, all of Denver. Two sons, Michael and Paul of Denver also survive, as does a sister, Sister Frances Bechtold, S.L.

* * *

Charles Bennett Wills, MD, formerly of Denver, died Tuesday, December 14, 1976, in Sun City, Arizona, where he moved three years ago.

Born June 26, 1913, in Waupun, Wisconsin, he attended the University of Wyoming, then went on for graduate training to the University of Nebraska, where he received his MD in 1939. After an internship served at St. Luke's Hospital, he served a junior surgical residency, then a senior surgical residency at the Alameda County Hospital, Oakland, California. This was followed by a year at that same institution in his chosen specialty of proctology.

After three years of service in the Army during World War II he returned to Denver to practice. Ill health forced his retirement in 1973, and he moved to Sun City, Arizona.

He was a member of the Denver and Colorado state medical societies, and of the AMA. He was certified by the American Board of Rectal Surgery in 1950. He was a Fellow of the American College of Surgeons and a member of the American Proctological Society.

He was a Mason and a member of the First United Presbyterian Church. He held the academic rank of instructor in Surgery at the University of Colorado Medical School.

In 1939, he was married to Edna Jacobsen of Omaha, Nebraska, and she survives him. Also surviving are a son, John B. of Denver, and a daughter, Mrs. Nancy McMillan, Modesto, California, and a sister, Mrs. Shirley Ortiz of Denver.

Montana

Mr. L. Russell Hegland, retired Executive Secretary of the Montana Medical Association from 1950 to 1973, died January 22, 1977 in Billings. He was born July 13, 1908 in Great Falls, and was graduated in 1930 from the University of Pennsylvania. He was the first Executive Secretary of the Montana Medical Association.

David T. Berg, MD, Helena physician, died January 27, 1977, at the age of 79. Doctor Berg had been a member of the Lewis & Clark Medical Society and of the Montana Medical Association and of the American Medical Association. He received his medical education at the Washington University School of Medicine at St. Louis. His widow survives.

Earl C. Hall, MD, Laurel, Montana, died December 6, 1976 at the age of 87. Doctor Hall had been a member of the Montana Medical Association, the American Medical Association, and the Yellowstone Valley Medical Society since 1926. He was honored in 1971 as a member of the Montana Medical Association Fifty Year Club. He is survived by a son and two daughters.

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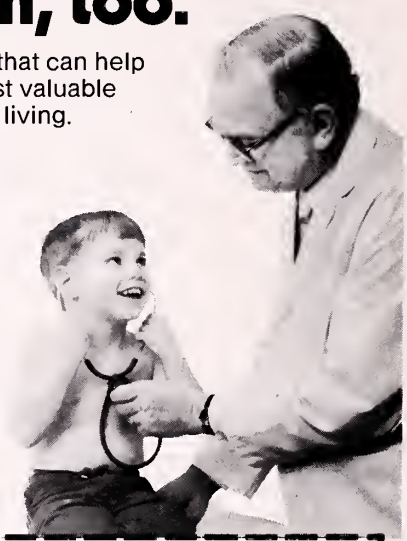

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MEETINGS

University of Colorado School of Medicine Continuing Medical Education

DENVER POSTGRADUATE INSTITUTE IN EMERGENCY MEDICINE

Denver General Hospital

May 2-7, 1977

* * *

INTENSIVE AND REHABILITATIVE RESPIRATORY CARE

University of Colorado Medical Center, Denver, Colorado

May 23-27, 1977

Contact: American College of Chest Physicians, 911 Busse Highway, Park Ridge, Illinois 60068.

* * *

FAMILY PRACTICE REVIEW

YMCA of the Rockies, Estes Park, Colorado

June 13-18, 1977

* * *

ORTHOPEDICS AND PHYSICAL FITNESS FOR THE FAMILY PHYSICIAN

The Given Institute of Pathobiology, Aspen, Colorado

June 25-29, 1977

* * *

PRACTICAL NEUROLOGY FOR THE INTERNIST AND FAMILY PHYSICIAN

The Given Institute of Pathobiology

July 11-13, 1977

* * *

GASTROENTEROLOGY: CURRENT CLINICAL TOPICS

The Gant, Aspen, Colorado

July 11-15, 1977

* * *

INTERNAL MEDICINE

YMCA of the Rockies, Estes Park, Colorado

July 18-22, 1977

* * *

PEDIATRICS

The Given Institute of Pathobiology

July 24-27, 1977

* * *

PATHOLOGY IN PEDIATRICS

The Gant, Aspen, Colorado

July 25-29, 1977

* * *

PEDIATRIC DERMATOLOGY

The Given Institute of Pathobiology, Aspen, Colorado

July 27-29, 1977

* * *

PATHOLOGY IN GYNECOLOGY AND OBSTETRICS

The Given Institute of Pathobiology, Aspen, Colorado

August 6-10, 1977

* * *

DISEASES OF THE KIDNEY AND RENAL FAILURE

The Given Institute of Pathobiology

August 10-13, 1977

Contact: Office of Postgraduate Medical Education, University of Colorado Medical Center, Container C295, 4200 East 9th Avenue, Denver, Colorado 80262

The Children's Hospital

PEDIATRIC CARDIOLOGY

Tammen Auditorium, The Children's Hospital,

1056 East 19th Avenue, Denver, Colorado

March 25, 1977, 8 am - 4 pm

* * *

PEDIATRIC ORTHOPEDICS

Tammen Auditorium, The Children's Hospital,

1056 East 19th Avenue, Denver, Colorado

April 22, 1977, 8 am - 4 pm

* * *

PEDIATRIC EAR, NOSE AND THROAT SYMPOSIUM

Tammen Auditorium, The Children's Hospital, Denver, Colorado

May 27, 1977, 8 am - 4 pm

Contact: **Betsy Block,** Department of Continuing Education, The Children's Hospital, 1056 East 19th Avenue, Denver, Colorado 80218, or Call: (303) 861-8888, Ex. 2280.

Montana Medical Association

1977 INTERIM MEETING

Colonial Inn, Helena, Montana

April 1-2, 1977

Department of Surgery, University of California, San Diego

MANAGEMENT OF TRAUMA

Sheraton-Harbor Island Hotel, San Diego, California

April 1-3, 1977

Contact: Nomi Feldman, Conference Coordinator, Box 705, La Jolla, California 92038.

The American College of Cardiology

VECTORCARDIOGRAPHY WORKSHOP

Scottsdale Hilton Hotel, Scottsdale, Arizona

April 13-15, 1977

NEW APPROACHES TO CARDIAC DIAGNOSIS

Fairmont Hotel, San Francisco, California

April 28-30, 1977

Contact: Mary Anne McInerny, Director, Extramural Programs Department, Division of Continuing Education, American College of Cardiology, 9650 Rockville Pike, Bethesda, Maryland 20014.

Institute for the Medical Humanities-University of Texas Medical Branch

FIFTH SYMPOSIUM ON PHILOSOPHY AND MEDICINE

University of California at Los Angeles, Los Angeles, California

April 14-16, 1977

Contact: Bernard Towers, MD, Center for the Health Sciences, University of California at Los Angeles, Los Angeles, California 90024.

Lovelace-Bataan Medical Center

COMMON PROBLEMS IN ENDOCRINOLOGY

Airport Marina Hotel, Albuquerque, New Mexico

April 15-16, 1977

Contact: Office of Medical Education, 5200 Gibson Boulevard, SE, Albuquerque New Mexico.

American College of Surgeons

TRAUMA-1977

Caesars Palace, Las Vegas, Nevada

April 17-20, 1977

Contact: John W. Batdorf, MD, 2300 Rancho Drive, Las Vegas, Nevada 89102.

Temple University School of Medicine

PRACTICAL CLINICAL CARDIOLOGY

El Presidente Hotel, Cozumel Island, Mexico

April 18-20, 1977

Contact: Albert J. Finestone, MD, Continuing Medical Education, Temple University School of Medicine, Philadelphia, Pennsylvania 19140.

American Academy of Pediatrics

2ND ANNUAL CONFERENCE ON NEONATAL-PERINATAL MEDICINE

Scottsdale Hilton, Scottsdale, Arizona

April 21-23, 1977

Contact: Donald Pickering, MD, c /o Conferences and Institutes, University of Nevada, Reno, Nevada 89557.

Stanford University School of Medicine

MANAGEMENT OF THE SURGICAL PATIENT

Mauna Kea Beach Hotel, Kamuela, Hawaii

April 30-May 7, 1977

Contact: Office of Postgraduate Medical Education, Stanford University School of Medicine, Stanford, California 94305.

American College of Surgeons

ANNUAL MEETING OF EL PASO CHAPTER

Four Seasons Motor Inn, Albuquerque, New Mexico

May 7-8, 1977

Contact: John B. Roberts, MD, 801 Encino Place, NE, Albuquerque, New Mexico 87102.

American Society for Clinical Oncology

SPECIAL MEETING

Denver Hilton, Denver, Colorado

May 16-17, 1977

Contact: Mrs. Jane Barnd, Charles B. Slack, Inc., Thorofare, New Jersey 08086.

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SOCIETY OFFICERS

Colorado Medical Society

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President: Carl H. McLauthlin, Denver.

President-Elect: William S. Curtis, Boulder.

Vice-President: James A. Philpott, Aspen.

Constitutional Secretary: Robert A. O'Dell, Aurora.

Treasurer: Kenneth R. Lovell, Colorado Springs.

Additional Trustees: William H. Call, Lakewood; Telford A. Davis, Durango; Abraham J. Kauvar, Denver; William D. McCrady, Denver; Joseph H. Poynter, Boulder; Ray G. Witham, Craig.

Delegates to the A.M.A.: Robert E. McCurdy, Denver, Dec. 31, 1977; Kenneth A. Platt, Westminster, Dec. 31, 1976; Joseph L. Kovarik, Denver, Dec. 31, 1977.

Alternate Delegates to the A.M.A.: Clyde E. Stanfield, Denver, Dec. 31, 1976; William Y. Takahashi, Boulder, Dec. 31, 1977; Leo J. Nolan, Lakewood, Dec. 31, 1977.

Speaker, House of Delegates: Robert F. Linnemeyer, Grand Junction.

Scientific Editor for Colorado, and Chairman of the Editorial Board, Rocky Mountain Medical Journal: Carl H. McLauthlin, Denver.

Scientific Editor Emeritus, Rocky Mountain Medical Journal: Douglas Macomber, Denver.

Executive Director, and Managing Editor, Rocky Mountain Medical Journal: Mr. Donald G. Derry, 1601 East 19th Avenue, Denver, Colorado 80218. Telephone: (303) 534-8580.

Wyoming State Medical Society

OFFICERS—1976—Terms of Officers and Committeemen expire December 31, 1976. Terms of office (with the exception of Delegate and Alternate Delegate to A.M.A.) expire at the end of the calendar year rather than at the Annual Session.

President: Patrick D. Nolan, Buffalo.

President-elect: James E. Stoetzel, Pine Bluffs.

Vice President: Kent T. Christensen, Casper.

Secretary: James R. Alexander, Torrington.

Treasurer: John C. Budge, Laramie.

Delegate to A.M.A.: Fenworth M. Downing, Sheridan.

Alternate Delegate to A.M.A.: John J. Corbett, Casper.

Speaker of the House: Theodore L. Johnston, Cheyenne.

Vice Speaker of the House: Ross J. Collie, Lander.

Immediate Past President: Donald B. Hunton, Cheyenne.

Scientific Editor for Wyoming, Rocky Mountain Medical Journal: Francis A. Barrett, Cheyenne.

Associate Editor for Wyoming, Rocky Mountain Medical Journal: Mr. Robert G. Smith, Cheyenne.

Executive Director: Mr. Robert G. Smith, P.O. Drawer 4009, Cheyenne, Wyoming 82001. Telephone (307) 635-2424.

Montana Medical Association

OFFICERS—1976-77—Terms of Officers and Committeemen expire at the Annual Meeting of the Year Indicated. Where no year is indicated the term is for one year only and expires at the 1977 Annual Meeting.

President: Colvin H. Agnew, Billings.

President-elect: James K. Cope, Forsyth.

Vice President: Robert P. Yost, Missoula.

Secretary-Treasurer: Paul R. Crellin, Billings.

Assistant Secretary-Treasurer: Jack L. Davis, Kalispell.

Delegate to the A.M.A.: Mark B. Listerud, Wolf Point.

Alternate Delegate to the A.M.A.: John R. Halseth, Great Falls.

Executive Committee: Colvin H. Agnew, Billings; James K. Cope, Forsyth; Robert P. Yost, Missoula; Paul R. Crellin, Billings; Jack L. Davis, Kalispell; David Gregory, Glasgow; Arthur C. Knight, Helena; Mark B. Listerud, Wolf Point; John R. Halseth, Great Falls.

Scientific Editor for Montana, Rocky Mountain Medical Journal: Gerald A. Diettert, Missoula.

Associate Editor, Rocky Mountain Medical Journal: Mr. G. Brian Zins, Helena.

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New Mexico Medical Society

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GASTROENTEROLOGIST needed for multispecialty group of 46 physicians. A unique opportunity to join a vigorous group in an excellent location. Write or call Paul V. Hoyer, MD, Medical Director, The Billings Clinic, P.O. Box 2555, Billings, Montana 59103, or (406) 252-4141. 177-7-2

LOCUM TENENS SERVICE: Young GP's will cover your practice 24 hous a day, 7 day week while you take your overdue vacation with the family. Salary or per centage negotiable. Write Box 177-3-3B c/o Rocky Mountain Medical Journal, 1601 East 19th Avenue, Denver, Colorado 80218. 177-3-3B

UROLOGY RESIDENCY: Unexpected opening. Second-year urology residency position available July 1977. Contact: T.A. Bordan, MD, Chief, Division of Urology, University of New Mexico School of Medicine, 2211 Lomas Blvd., NE, Albuquerque, New Mexico 87131. 377-1-1B

WANTED: DIRECTOR, FAMILY PRACTICE RESIDENCY. University of Wyoming, College of Human Medicine, Cheyenne, Wyoming. Responsibility for developing residency program in newly established statewide medical education program. Demonstrated interest in innovative medical education programs to help meet health care needs of the state. Experience in undergraduate medical education desirable. ABFP and their patient care experience desirable. Contact: David Holden, MD, Assistant Dean for Clinical Sciences, University of Wyoming College of Human Medicine, Box 3433 University Station, Laramie, Wyoming 82070.M/F. The University of Wyoming is an affirmative action/equal opportunity employer. 377-2-1B

WANTED: INTERNIST, board certified or qualified, for multispecialty group in SE Denver. General internal medicine, subspecialty training desirable but not necessary. Salary plus incentive first year, then partnership. Contact: Burton P. Golub, MD, 1919 So. University Boulevard, Denver, Colorado 80210, or call: (303) 744-2701. 377-3-1B

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377-9-1B

FAMILY PRACTITIONER/INTERNIST position openings in eastern Oregon rural community; Clinic site available; No investment needed; Solo or group practice; Progressive JCAH approved hospital; excellent business potential; four seasons; unlimited recreation. Write: Jim Mattes, Administrator, Malheur Memorial Hospital, 1109 Park Avenue, Nyssa, Oregon 97913, or Call: (503) 372-2211. 377-10-4B

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NURSE PRACTITIONER. Experienced, university-prepared, interested in working with Colorado physician(s) in private practice, or clinic setting, preferably in Denver or surrounding areas. For information, contact: Loretta Knauf, RNP, 840 So. Oneida St., # 209A, Denver, Colorado 80224, or Phone: (303) 355-7025.
377-12-1B

PHYSICIAN ASSISTANT seeks position with pediatrician. 1977 graduate of Western Michigan University with BS. AMA-approved. BS in child development and teaching. Four years as certified respiratory therapy technician, including special training for neonates. Write Kathleen Nitz, 709 W. Lovell, #2, Kalamazoo, Michigan, 49007. 377-13-1B

PRIMARY CARE PHYSICIAN'S ASSISTANT. Graduates from Western Michigan University August 1977 with BSM, AMA approved. Relocating in Colorado, seeks small group practice that could utilize a PA. BS - University of Wisconsin, four years experience Corpsman in USN. Write Donald Faulkner, 1605 W. North Street, Kalamazoo, Michigan, 49007. 377-14-1B

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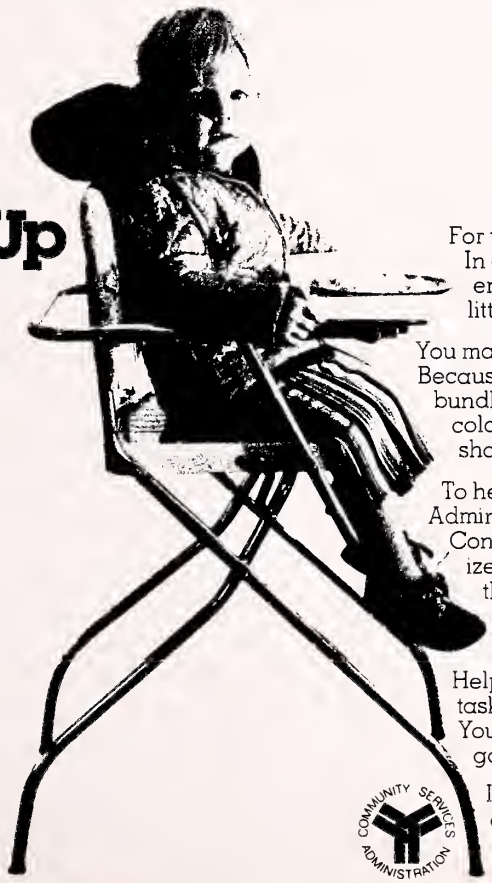
All Dressed Up for Dinner.



For the past two winters Andy dresses up for dinner. In a heavy coat and warm pants. It's not the way to enjoy a hot meal, but when the house is cold there's little choice.

You may think this is a special situation. But it's not. Because every year thousands of infants and adults bundle up in their own homes. They eat dinner in the cold and they go to bed in the cold. And it's a national shame.

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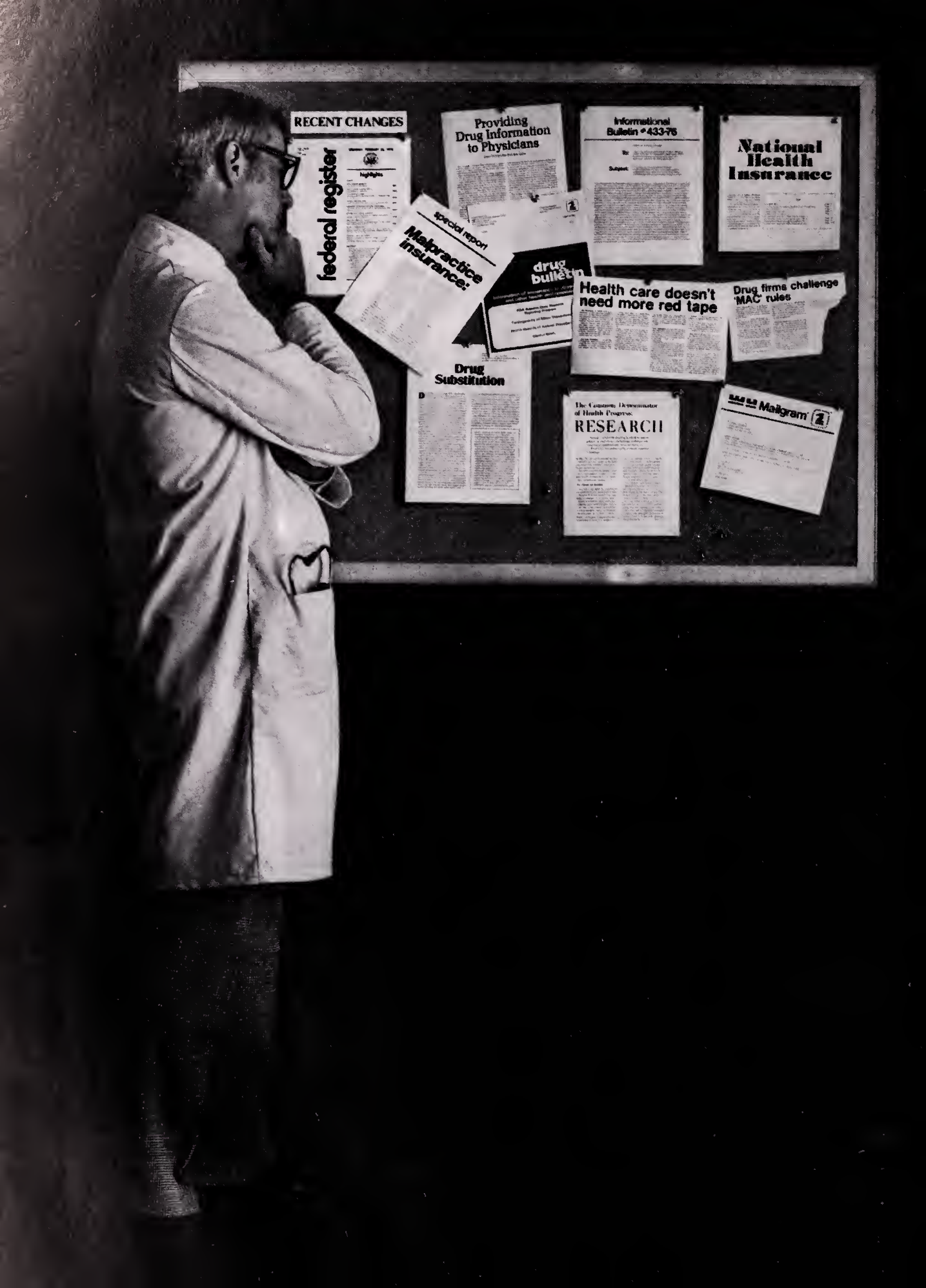
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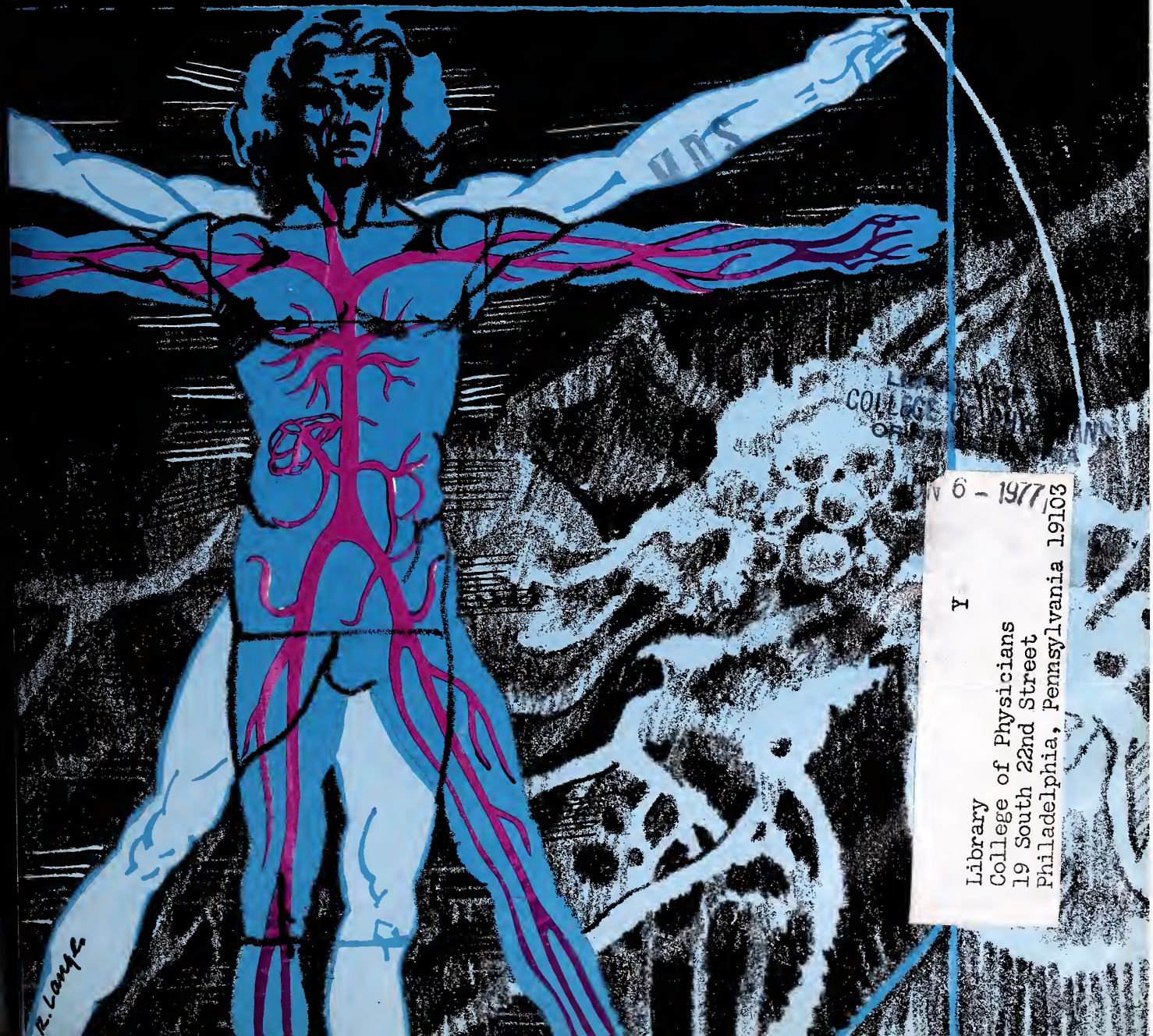
SPECIAL ISSUE:

The Clot



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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice, periodic blood counts and liver function tests advisable during long-term therapy.

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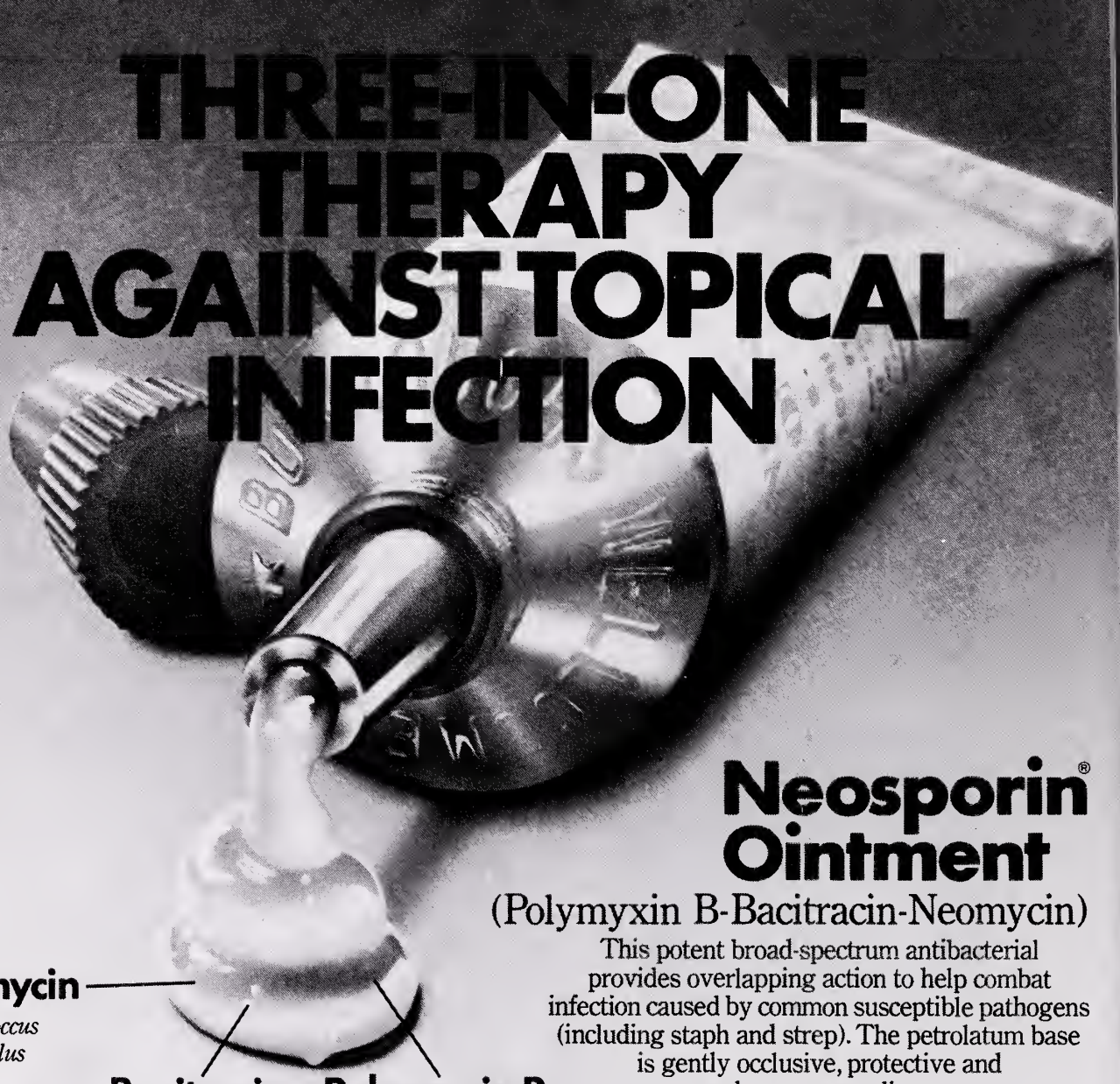

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OUR COVER, Richard Lange, graphic artist and illustrator who designed the cover to illustrate *The Clot* is newly arrived in Denver. Prior to his arrival here he free-lanced in Austin, Texas for five years and now maintains a studio at 84 S. Lincoln St.

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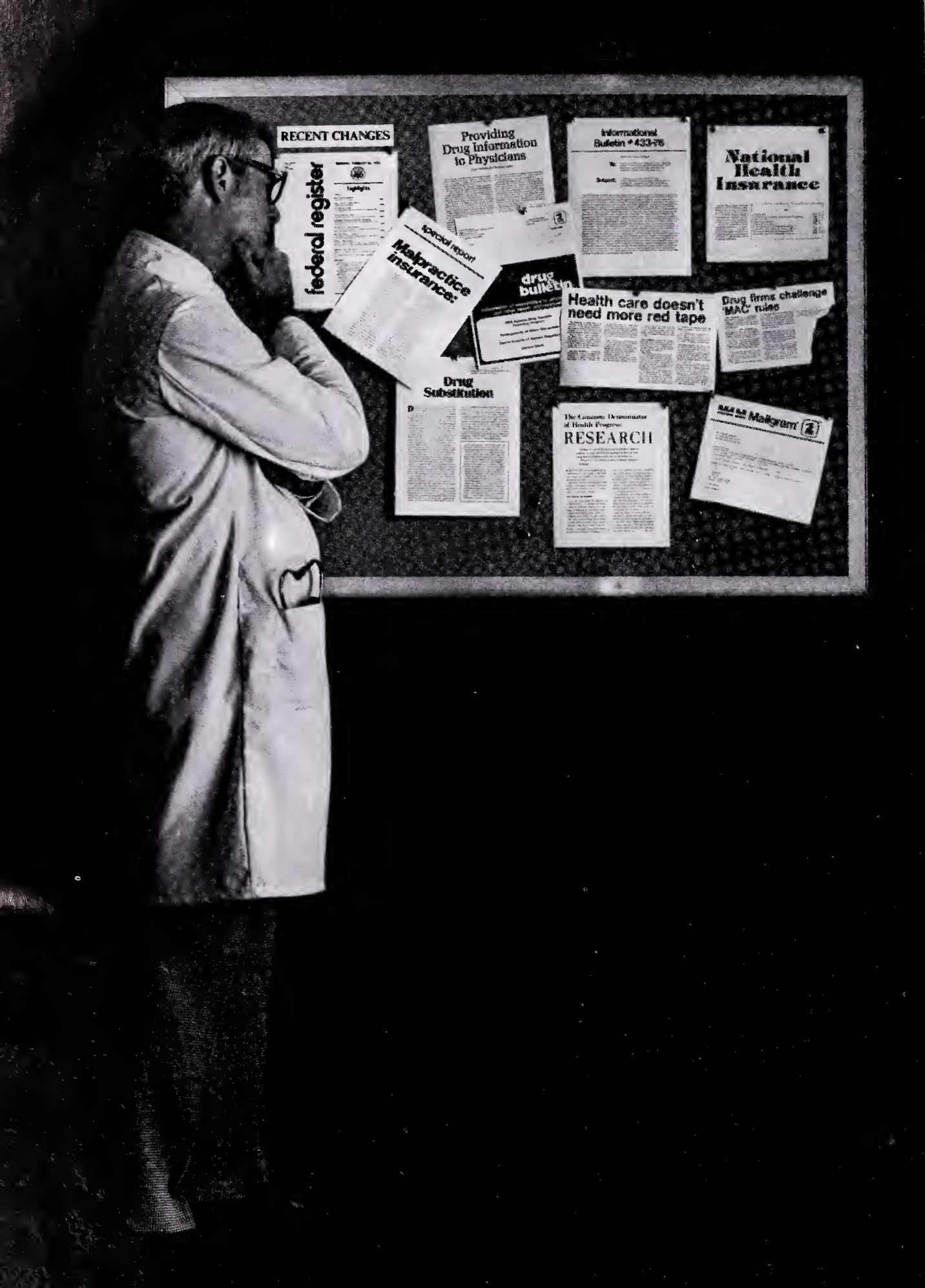
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Ironically, this dilution of physician control has taken place against a background of growing evidence that purportedly equivalent drug products may be inequivalent, since neither present drug standards nor their enforcement are optimal. In fact, the FDA itself says it has not enforced the same standards for hundreds of "follow-on" products that it had applied to the original NDA approvals. Thus physician control over patient therapy is being eroded with a risk that patients may be exposed to drugs of uncertain quality.

The major advertised claim for substitution is reduced prescription prices for consumers. Yet no documentation of any significant savings has been produced.

MAC Maximum Allowable Cost, MAC for short, is a Federal regulation designed to cut the Government's drug bill by setting price ceilings for drugs dispensed to Medicare and Medicaid patients. Unless the prescriber certifies on the prescription that a particular product is medically necessary, the Government intends to pay only for the cost of the lowest-priced, purportedly-equivalent, generally-available product. The effect of the program may be that elderly and indigent patients will be restricted to products which someone in Washington believes are priced right. Practicing doctors will have little to say about administration of the program, since Government will have absolute authority to make its choices stick.

The drug lag The future of drug and device research depends upon a scientific and regulatory environment that encourages therapeutic innovations. The American pharmaceutical industry annually is spending more than $1 billion of its own funds and evaluating more than 1,200 investigational compounds in clinical research. Disease targets include cancer, atherosclerosis, viruses and central nervous system disorders, among others. But there is a major barrier to the flow of new drugs to your patients: The cost of the research is more than ten times what it was, per product, in 1962; and whereas governmental clearance of new drug applications took six months then, it commonly consumes two years now.

The FDA needs adequate time, of course, to consider data. But it is equally clear that the present approval process contributes to needless delay of needed therapy. That's why the increased efficiency of the drug approval process is vital to all our futures.

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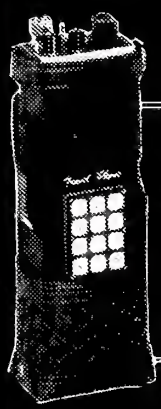

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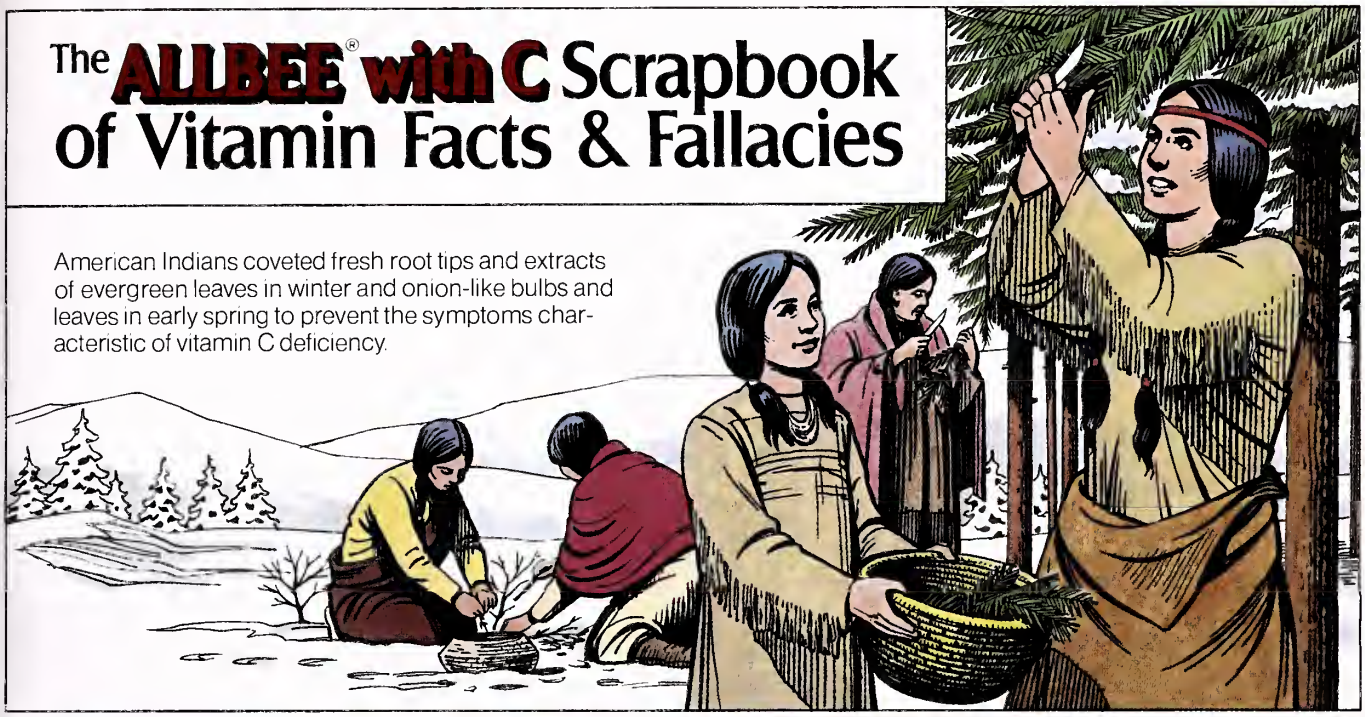

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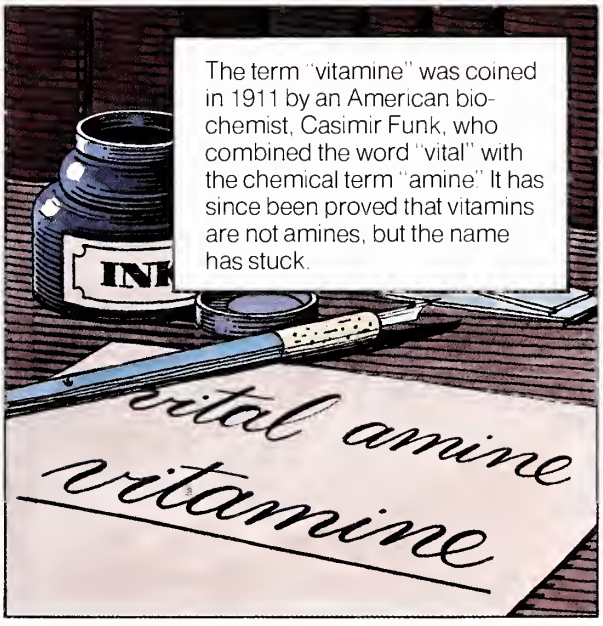
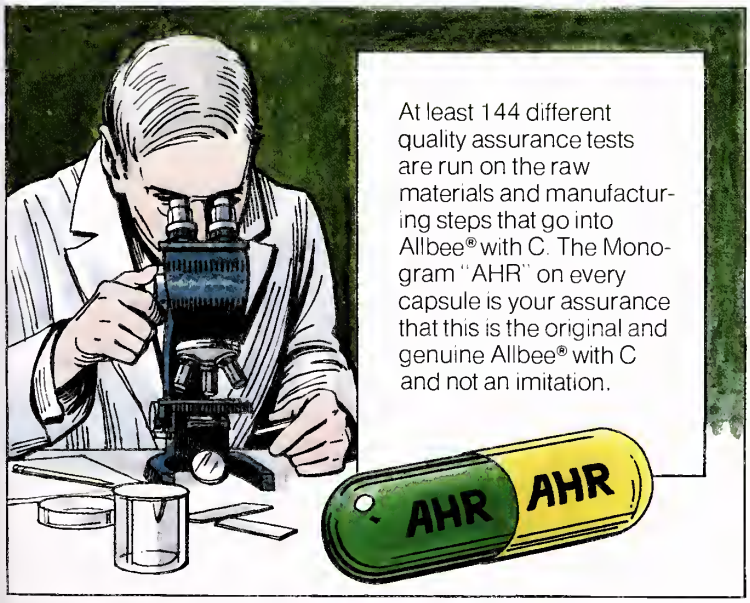


The term "vitamine" was coined in 1911 by an American biochemist, Casimir Funk, who combined the word "vital" with the chemical term "amine." It has since been proved that vitamins are not amines, but the name has stuck.



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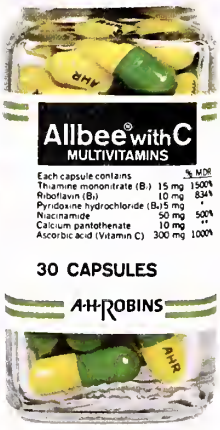

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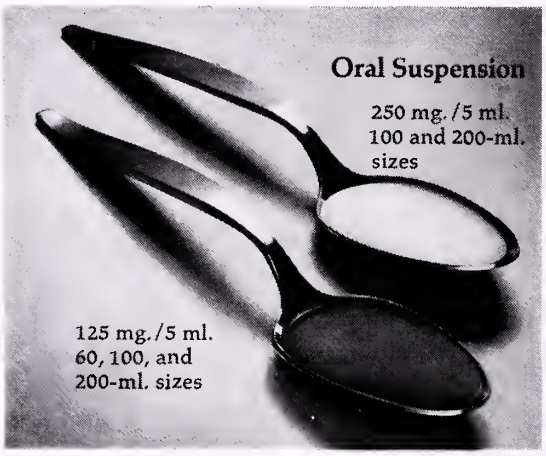

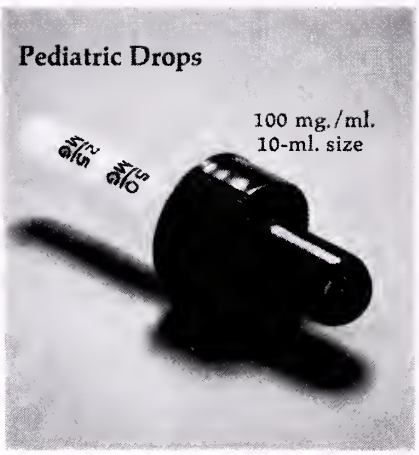

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To meet the demands and needs for health care there is continuing development of a wide variety of nonphysician providers. Definition and delineation of responsibilities of these providers and extenders is a complex and perplexing problem. Extension of activity of any health care provider can be done orderly and safely only when each individual of the provider group has standardized credentials of capability. Most people do not appreciate the effort and time required to develop an educational and training system which results in a uniform and standard product. It is important to remember how the present standardized education of the American physician came about.

The Flexner Report

At the start of the Revolutionary War, the training of most physicians in America was apprenticeship to an established doctor. For example, Dr. Joseph Warren, hero and patriot, had been the apprentice of Dr. James Lloyd, one of two physicians in Boston who had formal education in Europe. Joseph Warren was in turn the teacher of his brother John, who became the first professor of Anatomy and Surgery at Harvard Medical School, established in 1783. Formal education in Europe was based upon the apprenticeship but also included formal lectures by the ranking physicians and observation of these teachers on their rounds in the hospitals. Some of the early American physicians who had supplemented their education by a learning period in Europe on return to their native land would offer courses of lectures. The American medical school developed out of these essays in medical teaching.

In 1765 John Morgan, a physician involved in these lecture activities proposed to the trustees of the College of Philadelphia a professorship in the theory and practice of medicine. The chair was established for Morgan, and soon thereafter a professorship of anatomy and surgery was added for William Shippen. Thirteen years earlier the Pennsylvania Hospital had been established, Benjamin Franklin being instrumental in its organization and development. Thomas Bond, who had started the hospital, and had been teaching bedside medicine in it, became the first professor of clinical medicine in the new school. Shortly thereafter Adam Kuhn joined the school as professor of materia medica, and Benjamin Rush became professor of chemistry.

Thus the first American medical school was a part of an institution of learning and closely associated with a large hospital. Before the close of the century, three more similar medical schools had been started, one in New York in 1768 to later become Columbia, one associated with Harvard in 1783, and one at Dartmouth College in 1798. But the sound pattern of origin of these first medical schools did not last. The rapid expansion of the country with the inevitable decay of the apprentice system fostered the development of medical schools independent of the university. Between 1810 and 1840 twenty-six new medical schools, largely proprietary, sprang up, and thereafter, in the United States and Canada in a little more than a century four hundred fifty-seven medical schools were founded. One hundred-fifty-five of these schools survived to 1910, the year of the Flexner Report.

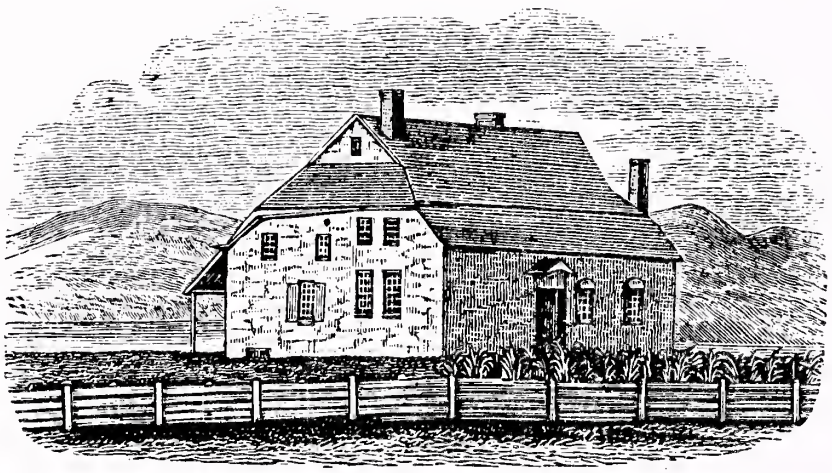
In about 1905 the trustees of the Carnegie Foundation faced a dilemma. They were entrusted with an endowment to be expended for the benefit of the colleges and universities of the

* *The Flexner Report on Medical Education in the United States and Canada, 1910.* By Abraham Flexner, reprinted by Science & Health Publications, 1740 N Street, NW, Washington, D. C. 20036.

United States and Canada. "It required but the briefest examination to show that amongst the thousand institutions . . . in North America which bore the name college or university there was little unity of purpose or of standards. . . Inevitably. . . . the scrutiny of the college led to consideration of the relations between the college . . . and the professional schools which had gathered about it . . ."* Colleges were found to have all sorts of relationships to their professional schools, many of the frailest texture in terms of responsibility, financial support, and educational conformity. During the period of development of these hundreds of schools the requirements of medical education had enormously increased. In 1908 the Foundation authorized a study of the schools of medicine and law in the United States. Mr. Abraham Flexner was engaged to study medical education.

There is no way to summarize the Flexner Report. It is a critique of medical education and a study of the process in America at that time so comprehensive and detailed that it is a marvel that it could be accomplished at all - and it was completed and composed in two years. There are two parts. Part I begins with a history of American medical education. Then Mr. Flexner describes the Proper Basis of Medical Education, the Course of Study, the Financial Aspects of Medical Education, the State Boards, and other related subjects with profound insight and logic to the degree that his analysis and proposals have remained the outline of medical education. Part II is a report of his findings of the one hundred fifty medical schools which he visited, each treated individually, with the deficiencies objectively and succinctly indicated, and with recommendations for improvements of the adequate institutions and dissolution of the inadequate.

The Council on Medical Education of the A.M.A. had been formed in 1904. After publication of the Flexner Report this Council and the American Association of Medical Colleges combined forces to develop standardized quality medical education in America - the modern medical school with standard requirements for admisssion, comparable didactic education, and uniform clinical learning in a hospital associated with the school, and the school a part of a college or university. The Flexner Report was the turning point, partly because of the influence of the Carnegie Foundation with its endowments, but primarily because of the thoroughness of the public exposure and the in depth logic of its proposals for medical education. Familiarity with the Flexner Report is essential for anyone engaged in defining the scope of activity of health care providers or in altering medical education in America.



Washington's Head-quarters, Newburg.

The clot:
A Symposium to Honor Kurt Nicolai von Kaulla, MD

Robert Chapman, MD, John Franks, MD, Roger Hamstra, MD, John Kurnick, MD, and Stephen Wallner, MD, Editor

In November 1974, Kurt von Kaulla returned to Germany. He had been on the staff of the University of Colorado School of Medicine for 21 years.

Kurt Nicolai von Kaulla was born in 1912 in Bornstarten, Germany, studied medicine in Bonn and Frieburg and received his degree in 1938. From 1939 to 1952 he worked in the research department in Frieburg and at the Geigy Pharmaceutical Company in Switzerland. He came to the United States in 1952 and for the next year was a research fellow at the New York University School of Medicine. He joined the faculty of the University of Colorado in 1953. He and his wife, Edith, brought modern concepts of coagulation to the University of Colorado School of Medicine and, in no small measure, to the entire Rocky Mountain medical community. His presence here shaped our thinking regarding coagulation problems and the management of bleeding and clotting disorders Scores of students and housestaff studied with him. His thorough evaluations of patients were known as "von Kaullagrams" and this term became shorthand for a coagulation workup done in his laboratory. During these years his professional development continued and today he is recognized as an international authority in the field of blood coagulation.

He is a member of numerous professional societies and the recipient of many honors, but the most notable thing about his career is his bibliography. It is virtually impossible to get an exact count on his bibliography because his output has been so profuse. It numbers somewhere in the neighborhood of 300 papers and books over his professional career to date. Many of these books and papers have been written in collaboration with his wife, Edith, a talented laboratory investigator.

In honor of Dr. von Kaulla, the Hematology Division of the Department of Medicine at the University of Colorado School of Medicine presented a postgraduate symposium in which we reviewed certain aspects of coagulation. The papers which follow are transcripts of this symposium. We hope to pay tribute to Dr. von Kaulla and his work in these papers. These reports are not intended to be all-encompassing reviews of the areas discussed, but rather we hope that the papers will serve to update the knowledge of the general physician, the person who first encounters clotting lesions in his daily practice.

From the Division of Hematology, Department of Medicine, University of Colorado School of Medicine, and the Denver Veterans Administration Hospital, Denver, Colorado 80220.

The clot:
The hypercoagulable state

Stephen F. Wallner, MD,* Denver

For this symposium, a brief review of hypercoagulability is a logical beginning. Dr. von Kaulla was instrumental in developing this field, and indeed, the majority of his work over the last decade has been with the development of concepts and tools for understanding the hypercoagulable state. Although this is a common and important area of medical practice, it is one about which we presently know very little. For example, Williams' *Textbook of Hematology* devotes 137 pages to consideration of bleeding disorders. There are eight pages of this text entitled "Thrombosis", and of these, all but one column of print has to do with the anticoagulant therapy of thrombotic diseases. Therefore, only one column in a 1500-page textbook of Hematology is devoted to the notion that there may be groups of patients who tend to form clots.

The potential clinical impact of hypercoagulability becomes apparent if one considers the range of human conditions in which clot formation may play some role. In most medical practices, patients with venous and arterial thrombotic and embolic disease are encountered daily. In addition, the clotting system appears to be important in the natural history of coronary and cerebral artery disease, not only when a clot is formed as a final event, but very likely in the actual generation of the atheromatous lesions. At the present time, however, this general area is truly an infant discipline. In most clotting states, our concepts of the development of the hypercoagulability are tentative and the pathophysiology is poorly understood. Many of the technics for evaluating patients are cumbersome and not routinely available, so that we are only beginning to develop methods to approach this area of human disease.

* Dr. Wallner is Assistant Chief of Medical Service, V.A. Hospital, Denver and Assistant Professor of Medicine, University of Colorado School of Medicine, Denver.

Classification of Hypercoagulable State

One can define two sets of patients who have the hypercoagulable state. The first group consists of those persons who have clinical, *in vivo,* evidence of hypercoagulability because they form clots. The second group is comprised of patients who, by one or more testing procedures, have evidence that their clotting system is more active than normal. These patients have the *in vitro* hypercoagulable state. The two groups must be considered separately although there is overlap in some instances. There are many patients who are clot-formers but who do not have *in vitro* evidence of a hypercoagulable state. For example, a number of patients form clots largely because of local venous stasis, and there are no currently available laboratory technics for detecting stasis; we may be able to discover hypercoagulability in more potential clot-forming subjects when our laboratory tools improve. Conversely, there are large numbers of patients who have the *in vitro* hypercoagulable state but who do not have a clinical clotting state when they are studied. At this time, we are usually unable to predict which of these subjects will clot. Clinical correlations are urgently needed.

The well-known hypercoagulable state that exists in women who are taking birth-control pills illustrates the problem. It is estrogen that causes hypercoagulability. Of all women taking birth-control pills, a significant fraction will develop some *in vitro* evidence of hypercoagulability but only a very small number will develop clinical hypercoagulability manifest by clot formation. We cannot at present accurately

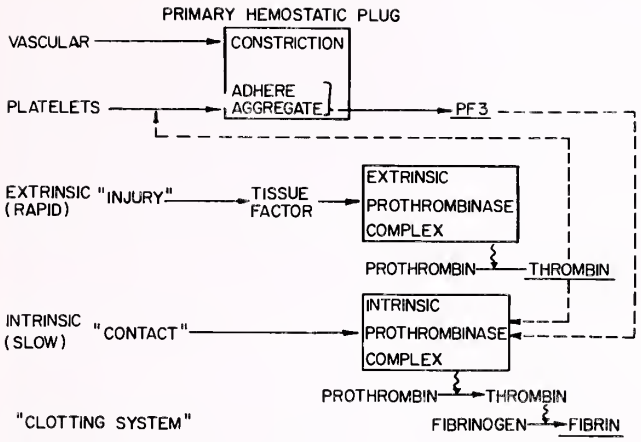


Fig. 1. Normal Hemostatic Pathways.

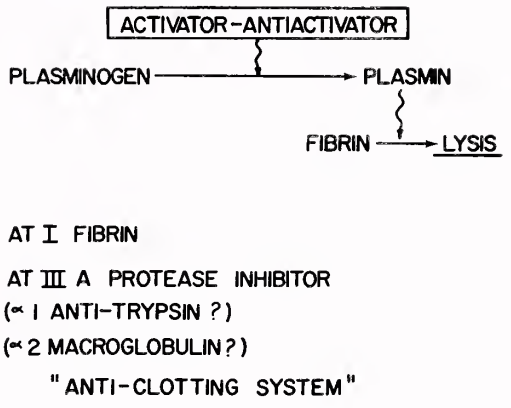


Fig. 2. Naturally Occurring Anticoagulants.

predict which patients will form clots. Dr. von Kaulla believes that the *in vitro* studies allow us to make some judgments on the severity of a hypercoagulable state and to make some predictions as to which patients may be at risk of clotting. In his first series of patients on estrogens he identified four women whom he felt showed marked evidence of hypercoagulability *in vitro*. He advised all four of them to discontinue the estrogens. Two did and two did not. Within weeks one of the two who had remained on the pills developed a serious pulmonary embolism and almost died and the fourth patient decided that she had better accept Dr. von Kaulla's advice and discontinue the pills. But this is only anecdotal evidence and prospective studies are needed.

Physiology of Clotting

A review of clotting physiology is necessary in order to develop some idea of how a hypercoagulability state develops. Fig. 1 shows the three major mechanisms which operate in an attempt to stop the loss of blood from injured blood vessels. The vascular phase participates in hemostasis because blood vessels can constrict to stop the flow of blood. We usually think of this in terms of capillaries and the fact that they are capable of closing down, but even large muscular arteries, when severed, can squeeze down to slow blood loss. The second contribution to overall hemostasis is made by platelets. Platelets do two things when an injury occurs. First, they stick to the severed vessel wall (adhesion) and to each other (aggregation) and form the "primary hemostatic plug". After platelets have aggregated they undergo a process called viscous metamorphosis and release a material called Platelet Factor III. The third contribution to hemostasis is the humoral phase which consists of the extrinsic and the intrinsic coagulation systems. The extrinsic system, which has the survival advantage of working rapidly, is based on a tissue factor (or tissue thromboplastin), a lipoprotein, which forms a complex with Factors VII, IX, X and calcium, and catalizes the conversion of prothrombin to thrombin. The amount of thrombin formed *in vivo* in the extrinsic system is relatively small, but the main end product appears to be these rapidly formed traces of thrombin. The intrinsic system is a slower system and is initiated by "contact." The exact sequence of events which follow "contact" is not important in this discussion. These details are widely argued. Dr. von Kaulla once remarked that the only thing that is certain about what happens is that it is very complicated. Ultimately, an intrinsic prothrombinase complex is formed, and this is capable of converting prothrombin to thrombin and then fibrinogen to fibrin. The intrinsic system produces thrombin in amounts sufficient to form fibrin clots. It is important to recognize the interactions between these systems. Obviously, the platelets are needed to produce Platelet Factor III, a material which is required for the formation of the intrinsic prothrombinase. Traces of thrombin formed by the extrinsic system contribute to the intrinsic system by hastening activation of Factors V and VIII. Thrombin is also efficient in causing platelets to aggregate. So the systems result in formation of a fibrin clot.

Since, in a sense, the clotting system is self-perpetuating, important mechanisms

have developed to keep it in check, once activated. Fig. 2 demonstrates the "anticlotting" system, which serves to stop clots from occurring and to confine fibrin formation to an injured area. Two different mechanisms are involved. The first is the plasminogen-based fibrinolytic system. Plasminogen is a serum protein which can be converted to plasmin under the influence of a material that is simply called "activator." Blood vessel walls are rich in a plasminogen activator substance which has been labeled "vascular activator." In addition, active Factor XII can convert plasminogen to plasmin. This is the factor activated by "contact" in the intrinsic system. It is peculiar that the factor initially responsible for intrinsic coagulation is also responsible, in part, for the eventual dissolution of the clot. The "activators" convert plasminogen to plasmin, a proteolytic enzyme with the property of being able to lyse fibrin. This system becomes active whenever fibrin is formed. The fibrinolytic system involves more than only lysis of a formed clot. As soon as fibrin is formed the fibrinolytic system will begin to destroy the fibrin strands. This is a progressively active system. It does not require a macroscopic clot to be activated.

The second anticlotting system is the naturally-occurring anticoagulants, collectively called antithrombins. These are materials with the capability of neutralizing thrombin and stopping the clotting system. Antithrombin I is fibrin. The clot itself has the property of adsorbing and neutralizing thrombin. Antithrombins II and III are closely related and may be identical. Dr. and Mrs. von Kaulla were instrumental in developing concepts and testing methods related to antithrombin III. A number of the active clotting factors are serine proteases, including thrombin and active Factors XI, IX, and X. Antithrombin III is a protease inhibitor and is capable of neutralizing these proteolytic enzymes. Most authorities believe that the most important naturally-occurring anticoagulant is antithrombin III. Heparin is a cofactor for antithrombin III and markedly potentiates its activity as an antithrombin. It appears likely that the anticoagulant activity of heparin is mediated through antithrombin III. Other plasma protease inhibitors which may play roles in modulating coagulation include "serum alpha 1-antitrypsin" and alpha 2-macroglobulin.

With these physiologic principles in mind, it should be clear that a hypercoagulable state can develop because of either an increase in the activity of the clotting system or a decrease in the activity of the anticlotting system, or both. For example, if the number of platelets is increased, or if they are being damaged, as in microangiopathic processes, Platelet Factor III may be released in excess, and the system tipped in the direction of fibrin formation. If tissue injury occurs, tissue factor may be released resulting in a "push" towards hypercoagulability. This mechanism may account for the important clinical clotting state seen in many patients with malignancy.

Systemic vasculidities may result in abnormal vessel surfaces activating the intrinsic system and leading to the formation of clots. Of interest is the fact that a hypercoagulable state can be detected *in vitro* in some patients with serum lipid abnormalities. This is not too surprising since serum lipid abnormalities are due to lipoprotein abnormalities and tissue factor is a lipoprotein. It empirically makes some sense that if serum lipoproteins are increased, it is possible that they may also act as a tissue factor-like material. As we shall report below, deficiencies of antithrombins have been associated with clotting states.

Tests for Hypercoagulability

We will now consider some of the laboratory methods which have been developed to detect hypercoagulability *in vitro*. A prothrombin time shorter than control does not appear to be of any value in detecting hypercoagulability. However, short partial thromboplastin times (PPT) are not infrequently noted in patients with a clinically active clotting process. As far as the vessels are concerned, a skin biopsy may play a role in looking for abnormalities, but will be rarely indicated. Platelet function can be studied in two different ways. The first way is by an *in vivo* method called platelet survival, analogous to red blood cell survival. It is simply done by taking some platelets from a patient and labeling them with ^{51}chromium and seeing how fast the tag disappears. If the label disappears faster than normal, it indicates that the platelets are being consumed and implies an

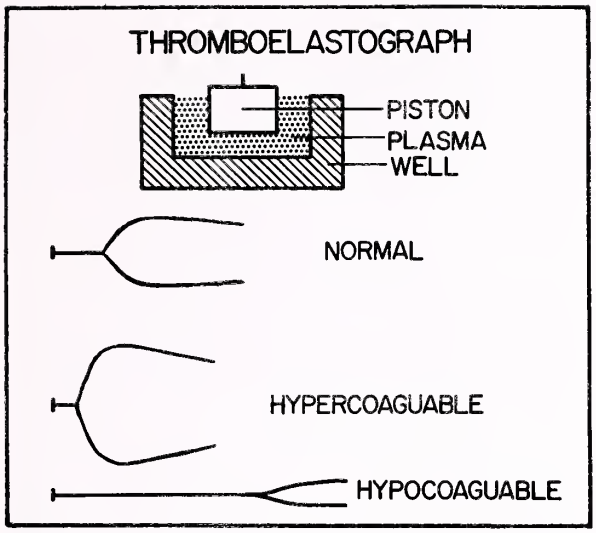


Fig. 3. The Thromboclastograph. The Upper Tracing is Taken from a Normal Subject, the Middle from a Hypercoagulable Patient, and the Lower from a Patient with Classical Hemophilia.

abnormality of the platelet-vessl interface and correlates with hypercoagulability. Short survival time does not necessarily imply an intrinsic platelet functional defect, since this result will be seen when normal platelets are consumed on an abnormal vessel structure, as in patients with mechanical heart valves.

In vitro tests of platelet function include studies of the ability of platelets to adhere and aggregate. Adhesivity refers to the platelet property of being able to stick to foreign surfaces. In the laboratory, adhesion is measured by passing platelet-rich plasma through a column of tiny glass beads and calculating the per cent of platelets which have adhered to the beads. The normal values are standardized in each laboratory.

Aggregation refers to the ability of platelets to stick to themselves and is measured by an instrument in which a light is passed through a platelet suspension. As the platelets aggregate, the light beam is scattered, the optical density of the system changes, and one obtains a measure of platelet aggregation. A further refinement of this method is to add substances which are known to speed platelet aggregation to the test platelet suspension. Such substances include collagen, thrombin, ADP, and epinephrine.

Three studies that are of value in detecting hypercoagulability in the humoral phases of coagulation are antithrombin III levels, thromboelastography, and thrombin generation. Antithrombin III can be directly measured in an *in vitro* system. The thromboelastograph is a simple machine in principle (Fig. 3). It consists of a piston fitted into a well. Citrated platelet-rich plasma is placed into the well and the piston is immersed in the plasma. The plasma is recalcified and coagulation begins. During the study the well rotates to-and-fro very slightly. If there is no fibrin connecting the well to the piston, the piston will not move. As soon as fibrin strands connect the well and the piston, it will begin to move. The piston is connected to an optical system which records how much it is moving relative to the well. The upper diagram in Fig. 3 is a normal thromboelastogram; the flat line represents the time it takes for fibrin formation to begin; the upslope represents the speed of fibrin formation, and the strength of the clot. Patients with hypercoagulability begin to form fibrin more rapidly; the upslope is faster and the depth of the curve is greater indicating that the clot which is formed is stronger, since more fibrin is formed. Finally, the lower panel shows a classical hemophiliac curve; the onset of fibrin formation is much delayed and proceeds very slowly, producing a very weak clot.

In the thrombin generation test, a tube of platelet-rich plasma is recalcified to start coagulation. At measured intervals, serial samples are removed and placed in a standard fibrinogen solution. The rapidity with which the fibrinogen clots is a measure of how much thrombin went into the tube, and reflects the amount of thrombin formed in the initial generation tube of patient's plasma. The more thrombin generated, the faster the fibrinogen will clot. Dr. von Kaulla devised this study. In the hypercoagulable subject thrombin is generated more rapidly and in greater amounts than normal, whereas the hypocoagulable patient shows delayed thrombin production, and the amount formed is much less than normal. The test is simple in concept, but because it is technically difficult to perform, it has not become popular among coagulationists.

Clinical Correlates

Few clinical correlates of the laboratory tests of hypercoagulablility exist. Very small numbers of patients are involved because not

many groups have been adequately studied. Disseminated intravascular coagulation will be discussed in a following paper. Another group which has been well studied includes patients with artificial heart valves. The platelet survival test has been used to evaluate these patients who are definite clinical clotters. They have an abnormally short platelet survival because they are consuming platelets too fast. As mentioned above, in this disorder the platelets are functionally normal. The survival is short because the platelets are laid down on the abnormal interface with the artificial valve. If these patients are treated with an agent designed to impair platelet function such as sulfinpyrazone, platelet survival is improved and there is a decreased incidence of clotting events. This is an important group of patients, since they serve as a prototype for the study of hypercoagulability: they are known clot-formers and an *in vivo* study correlates with the clinical clotting state; we know why they form clots; specific treatment directed at platelet function normalizes the *in vivo* hypercoagulability.

Another important group of patients currently being studied consists of those with coronary artery disease. These patients frequently have reduced platelet survival and reduced antithrombin activity. In this condition, however, it is much less clear as to what is "cart" and what is "horse". Some investigators believe that the clotting system is involved in the onset, and progression of atheromatous disease is focused on platelets. Other data suggest that the evidence of hypercoagulability is secondary to preexisting abnormal vessel surfaces. In any event, studies directed at interference with platelet function are in progress in a number of centers.

Some useful data have come from the *in vitro* studies of platelet function. The area of greatest interest is patients with transient ischemic attacks. In many patients it appears that these episodes are due to platelet emboli which originate on plaques in the great neck vessels. In some of these patients, evidence of increased platelet function has been found *in vitro*. This appears to correlate with the clinical propensity to ischemic episodes. A number of neurologists are treating these patients on a research basis with agents designed to inhibit platelet function. In addition, information is being obtained to see if there is some relation of platelet function to the cerebral vascular accident complex of diseases.

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Only limited data currently exist correlating clinical states with the studies of the humoral phase of coagulation. Women on birth-control pills often have a reduced antithrombin III level and accelerated thrombin generation, but clinical correlates are poor. Dr. von Kaulla has described hereditary antithrombin III deficiency in a large family. Many members of the family were subject to significant clinical clotting disorders, including pulmonary embolism and recurrent thrombophlebitis. This finding has been confirmed and a number of reports of families with this lesion have appeared recently.

The patients described above have abnormalities of specific studies. Unfortunately, the bulk of patients with hypercoagulability fall into diffuse and poorly understood groups. For these patients, Dr. von Kaulla has emphasized that a battery of studies is needed. It is known that many patients with cancer and systemic vasculitis are clinically hypercoagulable, presumably due to tissue factor excess. Again, we are in need of clinical cor-

relates so that we can predict from the *in vitro* studies which patients might form clots. This is a critically important issue since anticoagulation would be of value, but cannot be applied indiscriminately to large groups of patients. Furthermore, it is not known whether or not hypercoagulability cycles, or whether or not patients may be at more risk at one time than another. Hypercoagulability is an area deserving intensive study.

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The clot:
The bruising and bleeding patient

Roger Hamstra, MD, Denver*

Dr. von Kaulla's contributions to the management of bleeding and bruising patients, particularly his contributions to the management of the local hemophiliac population, have been substantial. Hemophiliac patients were regular visitors to his research laboratory where immediate help as well as hope for a better future was freely given. In recognition of this part of Dr. von Kaulla's activity, let us review an approach to the bruising and bleeding patient.

Our plan is to outline the history, physical examination, and stepwise logical moves which will avoid errors and lead toward, if not to, the proper diagnosis. Appropriate therapy will follow, perhaps as a joint effort with your consultant coagulationist. A majority of bleeding patients will have acquired conditions. Hereditary diseases often appear as new mutants or as mild disorders. About 25 per cent of hemophiliac patients come from families where the disease apparently has not been present before.[1]

The first matter is whether or not a problem of bruising or bleeding is present. Bruising and bleeding per se are not abnormal in the presence of substantial trauma. Inappropriate bruising and bleeding needs to be present. Table 1 provides four questions for each issue. A positive response to any one or more of these questions leads to further search. A negative response to each question provides strong evidence against a bleeding disorder.

Family history is important for boys and men, since classical inherited hemophilias are sex-linked. Though less rewarding, it should be included in the evaluation of a female. False positive responses are more frequent than true problems; patients always seem to

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TABLE 1

Bruise Too Easily?

New bruise each week?
Bruise without cause?
Bruises larger than 50¢ coin?
Bruises from injections?

Bleed Too Easily?

Bleed more than 30 minutes from a stick?
Bleed more than 1 hour from a bandaid cut?
Bleed a day or more after surgery?
Bleed a day or more after a tooth was pulled?

have a relative or two who have bled excessively. It is most helpful to get medical or laboratory documentation from relatives alleged to be bleeders, giving the laboratory a hint of the problem's nature.

The incidence of drug-induced minor bleeding and bruising tendencies is two to three times more common than an hereditary abnormality. This is particularly true in women, often being referred with a presumptive diagnosis of von Willebrand's disease or Factor XI deficiency. Usually, the drugs are platelet modifers or hemostatic plug modifiers, aspirin being the single most common agent. Laboratory evaluation proves to be normal when all aspirin-containing agents are avoided for at least ten days. Other drugs also modify platelet function.[2] Dipyridamole is well known for its platelet modifying properties, but others are less well known (Table 2). Agents such as glyceryl guaiacolate are much weaker than aspirin, but can become significant in an otherwise borderline patient. It is surprising how often the patient will be taking these drugs, complain of bruises, and not realize that the drugs play a role. Obviously, anticoagulants should be sought. In the hospital, incidental heparin administration may be important, such as through a heparin lock or the small doses of heparin used in IV solutions occasionally, to keep the indwelling line from clotting.

If all responses indicate no problem, there remains one additional maneuver to complete the negative screen, i.e., an assessment of platelet numbers. Thrombocytopenia can be silent and therefore either an estimate of platelet count from a peripheral blood smear,

TABLE 2

Drugs associated with Increased Bruising and/or Bleeding

Aspirin (in all its many forms) [3]
Sulfinpyrazone (Anturan T) [3]
Phenylbutazone (Butazolidin T) [3]
Dipyridamole (Persantin T) [3]
Penicillin G (high dose) [4]
Carbenicillin [5]
Dextran (low M.W.)
Hydroxychloroquine (Plaquenil T)
Nitrofurantoin (Furadantin T) [8]
Glyceryl Guaiacolate (Robitussin T) [9]
Clofibrate (Atromid--S T) [3]
Chlorpromazine (Thorazine T) [3]
Imipramine (Tofranil T) [3]
Amitryptline (Elavil T) [3]
Volatile general anesthetics [2]
Local anesthetics [2]
Heparin
Coumarin compounds

TABLE 3

Diseases Associated with Abnormal Bruising and/or Bleeding

Osteogenesis imperfecta
Hereditary Telangiectasia
Ehler-Danlos Syndrome
Margan's Syndrome
Pseudozanthoma Elasticum
Liver Dysfunction
Uremia
Giant Hemangioma

or a formal platelet count is needed. If the count of platelets is normal, no further search is needed. It is not our custom to order a laboratory coagulation screen if answers to questions are negative and the platelet count is normal. We consider this screen to be adequate for surgical candidates.

Comment is worthwhile on the issue of presurgical coagulation screening. A pattern of ordering a group of tests such as the prothrombin time, activated partial thromboplastin time, thrombin time, fibrinogen, bleeding time, and platelet count is present in some communities. If this is done in addition to careful medical team evaluation, it gives a small added degree of confidence. In our opinion, it is not better

TABLE 4

Laboratory Flow Sheet of Inherited Bleeding Disorders

Pattern		Screening Tests PT	APTT	TT	PCT*	Possible defect	Substitutions NP**	AS	AdP	Additional Tests
Hemostatic Plug	1	NL	NL	NL	AB	Thrombocytopenia	o	o	o	Platelet count, bleeding time (template)
						PF-3	o	o	o	Inosothin PCT NL; PF-3 assay
Extrinsic Clotting	2	AB	AB	NL	NL	X	+	+	o	Stypven time, specific assay for X
						V	+	o	+	I-V rich PT NL; specific V assay
						II	+	o	o	Specific II assay
Extrinsic Clotting	3	AB	NL	NL	NL	VII	+	+	o	Stypven time, specific assay for VII
Intrinsic Clotting	4	NL	AB	NL	AB	VIII	+	o	+	Definitive differentiation can only be established by specific factor assays
						IX	+	+	o	
						XI	+	p	p	
						XII	+@	p@	p@	
Thrombin Fibrinogen Reaction	5	NL	NL	AB	NL	Hypofibrinogenemia	+	o	+	Chemical and immunological assays of fibrinogen activity and migration
						Dysfibrinogenemia	+	o	+	
Stable Fibrin Clot	6	NL	NL	NL	NL	Clot is soluble in 5 m area. Corrects with small amounts (1%) of normal plasma				Clinical story of delayed bleeding, particularly a few hours or days after surgery, raises the issue of XIII deficiency

* Refers to prothrombin consumption performed without subsequent II assay.

** If normal plasma does not correct a test, platelet defect, an inhibitor, or hageman train is suspected (all Hageman trait patients may inactivate normal plasma, and apparent correction fail)
Legend: PT = prothrombin time; APTT = activated partial thromboplastin test; TT = thrombin time; PCT = prothrombin consumption test; NP = pooled normal plasma, diluted 1:1 with saline; AS = aged serum pooled, diluted 1:1 with saline; AdP = plasma adsorbed with either $BaSO_4$ or $Al(OH)_3$, diluted 1:1 with saline; + indicates correction; o indicates no correction; p indicates partial correction; @ indicates that Hageman plasma may inactivate any source of XII.

From: Handbook of Hemostatis by Thomas Zuck, M.D., LTC, MC, Fitzsimons Army Medical Center, Denver, Colorado (1973 Revision unpublished).

medical care, although one's posture in a medical-legal arena may be more comfortable. Too often, the laboratory becomes a substitute for evaluation at the bedside, particularly in a busy surgical sub-specialty practice. We have more confidence in the patient's past behavior than in laboratory tests. Normal laboratory tests can still be associated with abnormal bleeding and bruising and never guarantee freedom from the complication of surgical bleeding.

If the platelets are decreased from normal, investigation leads away from coagulation problems toward bone marrow and platelet consumption diseases. Abnormal cells on the peripheral blood smear or absence of normal blood cells points directly toward the bone marrow. Pure platelet deficiency usually is due to increased destruction, rather than decreased production, immune mediated thrombocytopenia being most common.

When platelets are present in normal numbers, but the patient has a bruising or bleeding problem by history, search for specific signs of a few unusual diseases associated with bleeding is appropriate (Table 3). Patients with these disorders are not often found, but failure to look for the problem almost always is associated with failure to recognize. With liver and renal disease, a complete coagulation screen may still be worthwhile. The coagulation laboratory and a consultant should be called, if the patient's problem is not resolved at this point. There is no single test to screen for the problem behind a bruising or bleeding patient. A battery of tests is needed, and often repeat studies are helpful, particularly in complex situations as-

sociated with fever, sepsis, neoplasm, drugs, liver and renal dysfunction. A good laboratory screen will evaluate five areas: hemostatic plug, extrinsic clotting, intrinsic clotting, thrombin-fibrinogen reaction, and clot stability (Table 4).

If the coagulation screen is normal, further efforts are not required and usually will not be productive. Surgery is reasonably safe. There remains a concern, however, because the patient's past behavior remains the most reliable screen and abnormal bleeding is occasionally encountered in a patient with normal laboratory studies. With unusual bleeding , abnormalities of the screen can be evaluated further to detect the precise mechanism (Table 4). Bleeding episodes and surgery are best managed with coagulation laboratory support, ready blood bank facilities, and involved interest from the consultant coagulationist.

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The clot:
Disseminated intravascular coagulation

John J. Franks, MD,* Denver

In organisms which have developed a circulatory system, an immense advantage is obtained by having a circulating fluid which can coagulate quickly and locally when the integrity of the vascular system is disrupted. This property is achieved, however, at the cost of great complexity, and as we know from many contemporary examples, complex things often go wrong. When we consider the coagulation system, it is not surprising that there is an occasional malfunction. What is surprising is that it does not fail more often than it does.

The terminal clotting factor in our highly complex, precariously evolved coagulation system is fibrinogen. On exposure to thrombin, fibrinogen loses peptides and forms long strands which, under the influence of fibrinoligase, or Factor XIII, become markedly insoluble. Collections of intertwined fibrin strands block holes in vessels, thereby inhibiting blood loss—the main physiologic function of stable fibrin. A secondary function

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of stable fibrin may be to provide a matrix for wound healing.

Malfunction of this terminal portion of the coagulation system can result in solidification of fluid plasma within the circulation produced by intravascular clotting. Depending upon the location of the fibrin so formed, this malfunction can be a disastrous and often fatal event. A complicating secondary event is the activation of the fibrinolytic or plasmin system with resultant hemorrhage. Plasmin lyses fibrin, which is a protective effect, but it also lyses fibrinogen and other clotting factors, and this adds to the consumption of factors and to the often grave hemorrhage associated with acute intravascular clotting. This linkage of inappropriate intravascular fibrin formation with excessive fibrinogenolysis is the hallmark of the syndrome of intravascular coagulation (IVC).

The following discussion of IVC will (1) describe the pathophysiology, (2) classify it into acute, subacute and chronic states indicating some of the precipitating causes, (3) describe diagnostic approaches to the clinical problem of IVC, and (4) briefly discuss treatment.

Pathophysiology

In disseminated IVC the release or activation of procoagulants by an initiating disease process, such as infection, malignancy or abruptio placentae, results in activation of the coagulation cascade. Accelerated consumption of fibrinogen, Factors II, V, and VIII, and platelets is accompanied by fibrin formation with deposition of fibrin in the microcirculation of the kidney, brain, lungs, and other organs. Fibrin deposition produces ischemia and frequently red cell damage with subsequent hemolysis. The characteristically distorted and fragmented red blood cells apparently result from passage through fibrin strands in the microcirculation. An important consequence of fibrin formation is the release of plasminogen activator from the anoxic vascular endothelium. The plasmin thus formed lyses fibrin polymer releasing fibrin degradation products; specifically fragments X, Y, D-dimer and E. This process is so efficient that despite a high rate of fibrin formation it is difficult to find even small thrombi in the vessels of patients who die with IVC. Plasmin also results in the lysis of circulating fibrinogen to produce fragments X, Y, D-monomer and E, and these have several anticoagulant effects. They have a direct antithrombin effect; they inhibit platelets; and they block polymerization of fibrin. Moreover, they form stable complexes with fibrin monomer. Inhibition of polymerization results in unstable fibrin monomer clots. Unstable clots also result from the coagulation of fragment X, which is clottable by thrombin. The activation of plasminogen is perpetuated by all these processes, and these two linked cycles of disaster continue unless the initiating event can be removed or, less successully, unless the consumption of procoagulants can be partially blocked by the paradoxical institution of heparin therapy in an often-bleeding patient.

The clinical and laboratory consequences of acute disseminated IVC can be seen in the purest form in experimentally produced IVC, achieved by slowly infusing thrombin into an experimental animal. Clinically, one usually sees increased blood pressure, bradycardia, tachypnea, cyanosis, convulsions, increased peristalsis, urination, defecation with bloody diarrhea, and oozing from wounds and needle-puncture sites. The laboratory manifestations of experimental IVC are decreases in fibrinogen, platelets, and Factors II, V, VIII, and XIII. Fibrinolysis can be observed by *in vitro* testing, and by increased consumption of plasminogen. The fibrin degradation products, as already mentioned, inhibit fibrin polymerization, have an antithrombin effect, and form an unclottable complex with fibrin monomer. In addition, there may be decreased platelet aggregation.

Classification and Diagnostic Approaches

The list of clinical conditions that may be complicated by IVC is continually growing, and the classification is arbitrary with a great deal of overlap. One category of IVC is initiated by what has been termed stagnant blood flow. The classic example is the Kasabach-Merritt syndrome, or giant hemangioma. Other conditions of stagnant blood flow may produce coagulopathies, congestive heart failure being chief among them. A second category of IVC syndromes is initiated by the release of thromboplastic substances. These can be further divided into several subclassifications. Obstetrical complications

include abruptio placentae, retention of a dead fetus, and amniotic fluid embolism. Hemolytic processes also release thromboplastic substances, e.g., transfusion of incompatible blood, fresh water drowning, immune hemolysis, extracorporeal circulation, march hemoglobinuria, and snake venoms of the specific hemolytic type. Tissue damage due to massive trauma, extensive burns, transplant rejection, and heat stroke comprise another subgroup. Another subgroup is neoplastic tissue necrosis, and we have evidence from our laboratory and from the work of others that neoplastic tissue may not have to be necrotic, in that there is a specific procoagulant produced by many tumor tissues. Fat embolization and snake venoms which have inherent thromboplastic activity are also included as a subgroup of this category. Finally, there is a group of IVC snydromes produced by mechanisms that are not understood. IVC is a complication of many infections, particularly gram negative sepsis.

Many of the entities already discussed can lead to an acute fulminant process of disseminated IVC which may terminate with the death of the patient within hours, unless interrupted by successful treatment. There are, however, subacute and chronic forms of IVC lasting days to weeks or even longer. These are becoming increasingly well recognized both as pathologic entities and as contributors to the pathophysiology of the primary underlying disease. The classification of IVC into acute, subacute, and chronic categories is arbitrary, and there is always considerable overlap. Chronic IVC differs from the acute form in several important respects. As in the acute form the coagulation system is activated, fibrin is formed, and secondary fibrinolysis develops. However, coagulation does not proceed at a rate rapid enough to deplete the system of coagulation factors, and instead there may be a paradoxical rise in their level so that one may even see hyperfibrinoginemia and increased levels of platelets. This is presumably because of an increased synthetic stimulus.

The best, and perhaps the only sure way to diagnose chronic IVC is with the use of carefully prepared homologous or, in man, autologous radioiodinated fibrinogen. When radioiodinated fibrinogen is injected into a normal man or animal, its behavior is predictable and consistent. After injection, plasma radioactivity disappears exponentially. Whole body radioactivity also disappears in an exponential fashion, and one can obtain whole body counts by placing the animal or patient in a whole body counter or by collecting urines and subtracting what was excreted from what was given. In the patient or animal with chronic IVC the picture is quite different from normal. Again, the plasma decays in an exponential fashion, but its rate of decay is much more rapid, indicating an increased rate of fibrinogen turnover. The tumor-bearing animals and patients whom I have studied all have high levels of fibrinogen so that there is an enormous increase in the rate of production and consumption of fibrinogen. In addition, the whole body radioactivity curve is less steep than the plasma radioactivity, because fibrin is being retained in the patient's body either as small clots distributed throughout the vascular system, or more commonly as clots around the neoplastic process. This process can be reversed by treating with heparin, in that the rate of disappearance of fibrinogen from the plasma can be retarded, and the whole body radioactivity and the plasma radioactive curves can be made parallel again if sufficient heparin is given. This strongly indicates that we are looking at a process of fibrin formation. It should be emphasized again that the clotting factors including platelets and fibrinogen are often increased in what we call chronic IVC, and that only by observing the rate of disappearance of one or more of these factors can diagnosis be made.

Labeled fibrinogen also provides the best means of diagnosing subacute IVC, which is defined as a process occurring at a rate sufficient to deplete or lower the levels of at least some of the coagulation factors. Hemorrhage and/or thrombosis may develop. The most characteristic example of subacute IVC is the Kasabach-Merritt syndrome.

In acute IVC, it is difficult to use labeled fibrinogen because often the interpretation of isotope technics is complicated by massive hemorrhage and by treatment. We must use indirect tests. Let us review once more the general laboratory picture in acute IVC. As a result of the consumption of platelets and other factors, there has been virtual conversion of the patient's plasma into something

very much like serum. There is a poor quality of clot formed. Fragmented red cells may be present. There is increased plasmin activity as evinced by rapid *in vitro* clot lysis. Products from the splitting of fibrinogen and fibrin are present, and they anticoagulate by interference with the remaining procoagulants. Finally, there is the effect of plasmin on fibrinogen and on Factors V and VIII.

How do we evaluate acute IVC? There are a number of useful screening tests which are usually available in a general hospital and which can be performed rapidly. The peripheral smear is an extremely useful examination, as nearly always in hematology, and the presence of fragmented red blood cells and decreased platelets should increase the suspicion of IVC. A low platelet count supplements examination of a peripheral smear. Fibrinogen level as measured by heat precipitation will be decreased. The prothrombin time, which is a measurement of not just prothrombin, but also of Factors V, VII, X and fibrinogen is a useful screening test, and will be prolonged. Another useful test is the activated partial thromboplastin time which measures the intrinsic system and the final common pathway, and this will also be prolonged. Thrombin time will also be abnormally long because of the low fibrinogen, and serial thrombin time will be progressively prolonged because of the antithrombin activity of the fibrin degradation products. Assay of Factors II, V and VIII would be expected to yield low results, particularly if the partial thromboplastin time is abnormal. Direct assay for fibrin split products by such methods as latex agglutination will be positive. Clot observation yields information about clot retraction during the first couple of hours. Clot lysis is considered abnormally rapid if it occurs in under twenty-four hours. There is also a rapid plasminogen screen available in some laboratories. A word of caution: there is no one test that is definitive in the diagnosis of IVC. Perhaps the most useful tests are measurements of platelets, fibrinogen, fibrin split products, prothrombin time and partial thromboplastin time.

Treatment

The principles of treatment are simple, but their practice is often terribly difficult. One should attempt to stop the initiating cause. Shock must be treated by giving fluids, blood, oxygen and bicarbonate. The only specific treatment is heparin and one must decide if this will improve or worsen the situation. One should never use the antifibrinolytic agent epsilon aminocaproic acid without prior heparinization of the patient, and then only if there is solid evidence of extreme activation of the fibrinolytic system. Finally, all tests and clinical observations must be repeated at frequent intervals in order to prepare for the rapid changes that occur in IVC if one is to rescue the patient from this desperate situation.

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The clot:
Idiopathic thrombocytopenic purpura

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Idiopathic thrombocytopenic purpura (ITP) is a disease of unknown cause, without known precipitating incident, wherein an antibody-mediated mechanism results in the destruction of platelets, and only platelets, primarily, but not exclusively, in the spleen. All these confusing modifiers make ITP of necessity a diagnosis of exclusion. We must, therefore, systematically examine and rule out the other causes of thrombocytopenia (Table 1).

Differential Diagnosis

As with red cells, one grouping of decreased circulating platelets results from decreased platelet production. The primary reason for decreased platelet production is decreased megakaryocytes, i.e., the marrow is aplastic or is replaced by a neoplastic process such as leukemia. The second reason for decreased platelet production is a direct toxic effect, as occurs for example with chemotherapeutic agents and includes an ethanol-related thrombocytopenia. Thirdly, there occurs decreased platelet production due to ineffective thrombopoiesis, which is seen in the same conditions and for the same reasons for which there occurs ineffective erythropoiesis. Impaired DNA synthesis in megaloblastic anemia results in ineffectivity of both erythropoiesis and thrombopoiesis. Parenthetically, it was once widely believed on morphologic grounds that ITP, with its abundant marrow megakaryocytes, represented a form of ineffective thrombopoiesis. It was later conclusively shown on the basis of studies with radioactively labeled platelets that in ITP there is a markedly shortened platelet survival, rather than ineffective production.[1] There exists, however, in a small number of patients, an ITP-like disease characterized by a normal platelet survival and idiopathic ineffective production of platelets, and resembling ITP clinically as well as morphologically in most other respects.[2] There are increased megakaryocytes in the marrow, but they are not making platelets, and this disease does not respond to steroids or splenectomy.

Another category of thrombocytopenias is represented by splenic sequestration. Although the spleen provides a very bad environment for red cells and the survival of red cells sequestered in the spleen is brief, twenty to thirty per cent of platelets normally reside there. In conditions where the spleen is enlarged there can be eighty per cent or more of the patient's platelets in the spleen at any given time. If enough platelets are sequestered in the spleen, there may be bleeding problems. Thrombocytopenia secondary to splenomegaly occurs most often in cases of portal hypertension, but is also seen in splenic infiltrating diseases such as lymphoma, agnogenic myeloid metaplasia, Gaucher's disease, and others.

Continuing the analogy with red cells, one cannot under ordinary circumstances lose sufficient platelets by bleeding to become thrombocytopenic without first exsanguinating. However, if blood is being replaced during a massive bleeding episode in the form of stored bank blood which does not contain viable platelets, one can produce a "wash-out" phenomenon and generate thrombocytopenia on that basis.

This brings us to the last category of thrombocytopenias which is, again by analogy to the anemias, that of increased platelet destruction. Mechanical damage to platelets

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TABLE 1
Classification of the Thrombocytopenias

I. Decreased platelet production
   A. Marrow aplastic or replaced
   B. Megakaryocyte toxins
   C. Ineffective thrombopoiesis

II. Splenic sequestration
   A. Portal hypertension
   B. Splenic infiltration

III. Volume replacement ("wash-out")

IV. Increased platelet destruction
   A. Mechanical damage to platelets
   B. Diffuse intravascular coagulation
   C. Immune damage to platelets
      1. Drug antibodies
      2. Isoantibodies
      3. Autoantibodies
         a. Postinfection
         b. Secondary to immunologic disorders
         c. Idiopathic thrombocytopenic purpura

by abnormal endothelial surfaces, as occurs in thrombotic thrombocytopenic purpura (TTP) and the hemolytic-uremic syndrome of children, can result in a consumption of platelets with platelet thrombi being formed, but without any significant amount of defibrination taking place. Lerner, Rapaport, and Metlzer[3] in an interesting report studied the clotting factors and fibrinogen levels in a patient with TTP who survived, and in whom there was good evidence that there was no consumption of clotting factors, although in many respects this disease does resemble diffuse intravascular coagulation, another important cause of thrombocytopenia. Finally, there are immune mechanisms for platelet destruction. A number of drugs can act as haptenes for antibodies and among them are quinine and quinidine, gold salts, and sulfa drugs (not just sulfa antibiotics but also the sulfonylurea antidiabetic agents and thiazide diuretics). There are isoantibodies, and analogous to erythroblastosis fetalis, isoantibodies to platelets have resulted in neonatal thrombocytopenic purpura. Isoantibodies have also been implicated in rare cases of accentuation of thrombocytopenia following the infusion of platelets. Autoantibodies are the last category. Autoantibodies have been seen after infections, especially after rubella and infectious mononucleosis, usually an abrupt and self-limited event. Autoantibodies have been seen secondary to established diseases of the imumune system, particularly chronic lymphocytic leukemia and lupus erythematosus, but all the collagen vascular diseases have been implicated in secondary thrombocytopenic purpuras. Finally, when there is nothing to which we can say that the immune thrombocytopenia is secondary, there is ITP.

Diagnosis

By history, the ITP patient probably notes increased bruising and bleeding, especially of the mucous membranes as manifested by tarry stools, excessive menses, or bleeding of the gums when brushing the teeth. The pertinent negatives are of great importance to this diagnosis of exclusion. There are no antecedent viral diseases, no offending drugs or known toxins in the patient's history, and no precipitating event. On the physical examination we have a patient who manifests purpura and petechiae. There is no splenomegaly, and no fever. Fever would point toward TTP, and a large spleen would point to sequestration. There should be no sign of red cell fragmentation on the peripheral smear. The white count is usually normal, as is the red count and everything else about the red cells, unless the patient has been bleeding enough to have developed anemia—of course, an iron deficiency anemia. Platelets are virtually absent from the peripheral smear. The platelet count is always below 100,000/mm^3, usually below 50,000/mm^3, and often below 20,000/mm^3. The tourniquet test, now largely obsolete, may be positive, and the bleeding time, although really indicated for qualitative rather than quantitative platelet problems, will be prolonged if it is done properly. The bone marrow classically shows increased megakaryocytes, but it is only fair to say that when we have looked at ITP marrows as unknowns we have usually assessed the megakaryocytes as adequate rather than increased. There is no way on purely morphologic grounds to assess how many platelets a megakaryocyte is producing or indeed, whether it is producing any platelets at all. Another pertinent negative for the marrow is the absence of leukemia or lymphoma.

There will be no abnormalities in prothrombin time and partial thromboplastin time, and there is no consumption of clotting factors. The Coombs' test is usually done and will be negative, although there is an entity called Evans' syndrome[4] in which there is a Coombs' positive hemolytic anemia in addition to thrombocytopenic purpura. The antinuclear antibody (ANA), to rule out lupus, should be negative. If performed, a platelet survival time will be greatly shortened, measured in minutes instead of a normal half-life which is in the range of five days. The latter test is preferably performed with autologous platelets to avoid unnecessary sensitization to allogeneic platelets, but this is often not possible technically when the patient's count is too low to enable one to harvest sufficient numbers of platelets for labeling.

Treatment

The best way to begin treatment is with prednisone or with another adrenal steroid at a dose equilvalent to about 60 mg/day. Whether one goes to 40 or 80 mg is not really of great importance, but it is usually found that if the patient does not respond within three weeks to 60 mg/day, he probably will not respond to an increased dose. There was a report from Cleveland in the late 1950's that in patients with ITP refractory to conventional steroid dosages, there may be transient beneficial responses to prednisone at 250-300 mg/day, sufficient to get the patient through an acute bleeding episode or to tide him over in the event of required emergency surgery.[5] We have little first-hand experience with this usage of large doses of prednisone, although our records include a referral patient unresponsive to prednisone at 250 mg/day. We were interested whether response to such massive steroid doses had any value in predicting response to splenectomy. In this patient there was no predictive value, since his response to splenectomy was excellent despite his failure on massive steroids. The problem of ITP patients who do not respond to the usual doses of prednisone requires further study. Although one cannot maintain patients on prednisone at 250 mg/day indefinitely, this dosage for a week or two followed by splenectomy could be tried whether or not the patient responds. If he does respond, the patient has the advantage of a normal platelet count at the time of surgery.

A patient who responds initially to steroids but relapses after discontinuation, or who requires maintenance steroids in order to sustain his platelet count, will almost always respond to splenectomy. Thompson and coworkers in 1972[6] demonstrated the value of steroid responsiveness in predicting response to splenectomy in 57 steroid-treated patients. Thirteen complete responses and 15 good responses were sustained for at least three months after discontinuation of the medication. Fifteen short-lived responses occurred, with counts rising to normal or near normal; however, these patients either required maintenance steroids or relapsed shortly after discontinuation of steroids. The remaining 14 failed to respond at all. The 13 with complete remissions all remained well without medication during one to eighteen years of followup. Of the 15 who had good responses, eight remained well at one to eighteen years of followup and seven eventually relapsed. Since 15 responses were short lived by definition, none continued in remission without drugs. Of 22 patients who relapsed after showing steroid responsiveness initially (i.e., 15 with short-lived response and seven with good response who relapsed), 18 underwent splenectomy. Of these, 16 remained well, one relapsed later after a brief response, and one made no response. This shows an almost ninety per cent complete response rate to splenectomy after initial responsiveness to steroids followed by relapse. On the other hand, patients who fail to respond initially to steroids have a response rate to splenectomy of only about 50 per cent. Thus, while failure on steroids does not necessarily predict a bad response to splenectomy, it lowers one to a category with a fifty per cent remission rate. By comparison, an initial steroid response conveys a thirty-seven per cent chance of not requiring any treatment after coming off steroids, and essentially a ninety per cent chance of remission following splenectomy should further therapy be required.

What about those patients who fail to respond to both steroids and splenectomy? In that group of patients we and others have traditionally tried immunosuppressive drugs such as cyclophosphamide, 6-mercaptopurine, and azathioprine. The results are somewhat

less than gratifying and difficult to interpret. Many patients manifest good responses at one time or another, but the responses often are not sustained. The platelet counts oscillate widely on the same dose of drugs, and may drop suddenly from 100,000/mm^3 to 10,000/mm^3. This variability always raises the question of whether the platelet-producing megakaryocytes have been destroyed by overtreating, or whether undertreatment has allowed the numbers of antibody-producing cells to increase, resulting in relapse. More recently, excellent results have been reported with vincristine in ITP resistant to steroids and splenectomy.[7] Our initial experience with this agent has also been gratifying, and we now believe that vincristine is the agent of choice in steroid and splenectomy resistant ITP.

There has been considerable debate about the long-term prognosis in ITP, since relapses have occurred after many years of complete remission.[8] There also has been dispute as to whether splenectomy is rational therapy for this disease, Dameshek having suggested that it may bring forth an occult lupus erythematosus.[9] Solid data supporting this concept is scanty, and such theoretical considerations should not deter recommendation of splenectomy in patients relapsing after, or unresponsive to, a three to four week trial of steroids. In the first group I would expect a complete response rate approaching ninety per cent, while in the latter I would not expect more than a fifty-fifty chance for remission. Since patients unresponsive to both steroids and splenectomy constitute roughly twenty to twenty-five per cent of the entire ITP group, it is helpful to have vincristine and the other cytotoxic immunosuppressive agents in reserve.

Finally, mention should be made of the use of platelet transfusions in ITP. While their use in several other forms of thrombocytopenia, epspecially those characterized by decreased platelet production, has proved valuable, in ITP the antibody which so dramatically decreases the survival of autologous platelets, has the same devastating effect on transfused platelets. Thus, platelet transfusions in ITP have little or no effect upon platelet counts. However, in the face of life-threatening hemorrhage, their use in desperation has been noted at times to be associated with beneficial results. Despite rapid removal from the circulation, it is probable that sufficient transfused platelets manage to get to the sources of bleeding to plug the holes. However, the prophylactic use of platelet transfusions in ITP should be discouraged.

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The clot:
Platelet Transfusion

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As previous discussants have indicated, the main form of treatment for bleeding disorders is replacement therapy. Platelet replacement in thrombocytopenic bleeding is now widespread in our country, and is growing. There is debate about the best way to prepare and use platelets in therapy, and further modifications in the coming years can be expected. I shall discuss preparation of platelets for transfusion, some of the indications for their use, amount to be administered, evaluation of response, and some complications which can occur in their use, and expected future developments in platelet transfusion.

Preparation

Platelets in the Denver community are obtained almost entirely from random blood donors essentially as a by-product of blood drawn for other purposes. For good platelet yields, the unit of blood must not be chilled before platelets are harvested. The blood bag is centrifuged lightly, enough to sediment the red cells but not the platelets. The bag is then placed in a squeezing device and the platelet rich plasma expressed into an attached satellite bag. This and subsequent manipulations are all done in a closed sterile system to avoid bacterial contamination. The only chance for contamination comes from the donor's arm at the time the blood is initially drawn. Nearly all the plasma is transferred, but we do try to avoid including any red cells or the buffy layer of white cells. Unfortunately, some red cells hang up around the outlet ports of the main bag and some red cell contamination cannot be avoided. In addition, lymphocytes tend to remain suspended in the platelet-rich plasma. The platelet-rich plasma is then centrifuged again, this time hard enough to pack the platelets in the bottom of the bag. The supernatant platelet-poor plasma can then be returned to the main bag to reconstitute platelet-poor whole blood or it can be transferred to another satellite bag for additional processing into cryoprecipitate or other protein fractions. All of the procedures up to this point are done at room temperature.

Some plasma must be left with the packed platelets to permit their storage for up to 72 hours. We have found we must wait 30 or 60 minutes before attempting to resuspend the packed platelets depending on whether acid citrate dextrose (ACD) or citrate phosphate dextrose (CPD) anticoagulant were used. Attempting to resuspend them sooner seems to tear up the platelets. If these units of platelet concentrate are to be used within 24 hours, 25 ml. of residual plasma is adequate, but if room temperature storage for longer is desired, between 35 and 50 ml. of plasma is left with the platelets to provide adequate nutriment and buffering. For room temperature storage, the platelets must be kept suspended, and for this the bags are kept on a rotator until shortly before use. If platelets are refrigerated for storage, rotation is not required and less residual plasma is needed.

Refrigerated platelets tend to aggregate and their life span in the patient is short. Nevertheless, they do seem to provide immediate hemostatic benefit in patients with thrombocytopenic bleeding. Room temperature stored platelets have a significantly longer life span in the patient with good hemostatic function although this function may not be quite as rapid in onset as that seen with refrigerated platelets. Because better survival of room temperature stored

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platelets gives a longer period of protection to the thrombocytopenic patient, all the Denver blood banks use this in preference to refrigeration. Even brief refrigeration is enough to alter platelet aggregation permanently, and it is therefore important not to follow the natural reflex of putting everything from the blood bank in the refrigerator if you want the advantages of room temperature stored platelets.

There are two ways to give platelets, either directly from the bag through a donor set containing a small filter, or by aspirating the contents of several bags into a syringe and then injecting them through a filtered intravenous line. Since four to five units of platelet concentration are usually given to an adult, it is probably better to give them directly from the bag. It is important to keep the platelets well mixed during administration and to infuse every bit possible so as not to waste any of them. Platelets will not pass through a micro-aggregate filter, so one of these filters must not be in the infusion line when platelets are administered.

Indications

Indications for platelet transfusions are not clear-cut. The patient's platelet count provides one guide. A count under 10,000/mm^3 is often, but not always, associated with serious bleeding problems. Some patients with counts of 5,000 have no trouble if they are kept at rest away from trauma. Counts of 10,000 to 30,000 are associated with mild to moderate risk, and above 30,000, risks of bleeding are low in the absence of trauma. The count should be higher if the patient faces surgery. In such patients, counts above 70,000 are desired. One exception would be the patient having splenectomy to alleviate excessive splenic destruction of platelets, since operation can be done with careful technic at much lower counts. Chronic thrombocytopenia generally produces less bleeding risk than does acute thrombocytopenia of the same severity, a situation explained by the fact that young platelets function better than old ones. The patient with longer standing thrombocytopenia will tend to shift his platelet age to younger levels, a shift indicated if the circulating platelets are larger than normal. Such patients are less in need of platelet transfusion than are those whose counts have dropped sharply in the last day or two.

In addition to the count, one should examine the patient for evidence of a bleeding tendency before deciding to transfuse platelets. Excessive ecchymosis around injection sites, spontaneous development of new petechia, epistaxis, and significant bleeding from the gums are other indications in making the decision, and active gastrointestinal bleeding or persisting heavy menstrual bleeding due to thrombocytopenia are indications for platelet transfusion. Hormonal suppression of menstruation in a woman with severe thrombocytopenia to prevent this potentially serious bleeding problem should not be omitted. Obviously if thrombocytopenia is complicated by other coagulation factor deficiencies, indications for platelet transfusion are increased.

The problem of whether or not to transfuse platelets to a patient who can be protected from surgical or other trauma and who is not currently bleeding seriously comes down to trying to assess the risk of developing intracranial hemorrhage, the one really lethal unpredictable complication of thrombocytopenia. Bleeding developing elsewhere can be treated by blood and platelet transfusion after it occurs, but bleeding into the closed intracranial space can lead to death if one waits for the serious headache and other signs to appear before giving platelet transfusions.

Amount

The number of units of platelets to give depends on the size of the patient, the desired increase in platelets, and the presence of already formed anti-platelet antibodies. One unit of fresh platelet concentrate contains about 10^{11} platelets. With 72 hour storage this amount will drop to about 5×10^{10}. In a child with one square meter body surface area one unit of platelets should raise the platelet count 12,000 to 15,000/mm^3. In an adult of twice this size, the increment would be half this much. In order to get an adult's count from 5,000/mm^3 to the safer range of 30,000/mm^3, four to five units of platelet concentrate are needed. If higher counts are needed in preparation for surgery other than splenectomy, more units will be required. If there are factors increasing the destruction of

platelets, especially fever, sepsis, or splenomegaly, more units will be needed. In the face of rapid platelet destruction due to antiplatelet antibodies or disseminated coagulopathy, transfused platelets are destroyed so quickly as to be useless.

Platelets can be given in three forms: (1) freshly drawn whole blood, (2) platelet-rich plasma, and (3) platelet concentrate. Freshly drawn whole blood was requested much more often than it is now. For the platelets to be effective, the whole blood must not be refrigerated before use, and it must be used before the various tests for safety, especially hepatitis tests, can be completed. Unless the patient is hemorrhaging, the red cell and plasma portions of the whole blood are unnecessary and in fact seriously limit the number of units that can be given without overloading the circulation. Platelet-rich plasma kept at room temperature can be used for patients with coagulation deficiencies in addition to thrombocytopenia, but again one is limited by the 200 ml. volume of each unit to relatively few units. In addition factors V and VIII in this plasma will deteriorate at room temperature. Platelet concentrate is the component preferred in most cases of thrombocytopenic bleeding because the small volume permits administering many units without worry about circulatory overload.

Response

Evaluation of the response to platelet transfusion is difficult. The platelet count should rise, usually reaching a maximum about one hour after transfusion. This hour's delay is apparently due to temporary pooling of the transfused platelets in the spleen, for delay does not occur in patients without spleens. Fever, sepsis, splenomegaly, and active bleeding will prevent much rise in count probably because they accelerate the consumption of the transfused platelets. Correction of the bleeding time has provided the only other objective indicator of response to platelet transfusion, but this often is not a practical test. There is the risk of subsequent scar formation and, especially in the patient who is also neutropenic, there is risk of infection. Clinical evaluation of the effect of platelet transfusion on the patient's bleeding problem, notoriously susceptible to subjective errors, remains the only other means to evaluate response.

Complications

Complications of platelet transfusions include infections and development of antiplatelet or anti-red cell antibodies. As with any blood component, except albumin and gamma globulin, there is the risk of hepatitis. Units are all tested for hepatitis B antigen, but occasionally, if the blood bank's platelet inventory is low, platelet units must be used before testing is complete. An additional hepatitis problem can arise, as described recently by Meyers, et al.[1] These investigators were collecting platelets by plasmapheresis, and, in order to get more than two units from each donor, they were putting needles through plastic bag seals. The needles had presumably become contaminated with hepatitis virus from a water reservoir used repeatedly for balancing centrifuge cups. This procedure led to several cases of hepatitis not only in patients receiving these platelets but also in the donors who had the residual cells and plasma returned to them during the plateletpheresis procedure.

Bacterial infection of platelet concentrates has been described and this problem could be accentuated by the warmer room temperature storage conditions. Further evaluations have shown no detectable viable bacteria in platelet units collected with good aseptic phlebotomy technic and strict use of sterile closed-system transfer procedures in the preparation of the platelet concentrate. Nevertheless, the occasional use of platelets stored beyond 72 hours because of inadequate blood bank inventory does carry at least a theoretically increased risk of bacterial infection.

So long as we use random unmatched donors for platelets, we can expect the appearance of anti-platelet (usually anti HL-A) antibodies in patients receiving platelet transfusions. This development does not ordinarily lead to clinically serious symptomatic reactions with additional platelet transfusions, but it does render such platelet transfusions ineffective. Antiplatelet antibodies promptly eliminate platelets from the recipient's circulation. This platelet-antibody reaction has been shown on occasion to lead also to temporary leukopenia, presumably on an immunologic basis. It will take a week or more for a previous unsensitized

patient to develop anti-platelet antibodies and refractoriness to platelet replacement therapy. Therefore random unmatched donor platelets will provide good replacement in short-term thrombocytopenic problems. If the patient has received blood transfusions in the past, or has had several pregnancies, there is the chance that anti-platelet antibodies are already present to interfere with response to platelet transfusion. In these situations platelet donors selected by HL-A type will be the only source for effective platelet replacement.
ment.

Units of platelet concentrate are marked to indicate the ABO and Rh type of the donor. In most situations this labeling may be ignored in selection of platelet units for transfusion. The unavoidable red cell contamination involves so few red cells that one need not worry about their hemolysis due to an ABO incompatability. Similarly the anti-A and anti-B present in the residual plasma of these units can be ignored because of the small plasma volume unless the patient is a baby or small child needing only one or two units for therapy. In these special circumstances plasma compatability should be observed. The main concern in situations of ABO and Rh incompatability is the possible subsequent development of antibodies against antigens contained on the contaminating red cells. Such sensitization is infrequent. Studies indicate that sensitization occurs in only about eight per cent of patients in which it is a potential. This low incidence probably reflects the poor immunologic status of patients needing platelets, since many of them have leukemia or cancer or are being treated with one or more immuno-suppressive agents. Nevertheless it is probably advisable to try to use platelets from Rh negative donors if the patient is a young Rh negative woman with potentially curable disease. Failure to observe Rh compatability in this circumstance could lead to the woman's developing anti-Rh $_0$ (D) with subsequent erythroblastosis in any baby she delivered who was Rh $_0$ (D) positive.

Future Developments

Future developments in platelet transfusion therapy will involve more availability and use of HL-A antigen identification for donor selection, improved methods of collecting several units of platelets from a single donor, and methods for longer platelet storage perhaps in the frozen state to permit better inventory control and to permit patients to store their own (autologous) platelets for their own future use. HL-A antigen matching seems to be important for satisfactory long-term effectiveness of platelet transfusion therapy. There is such a variety of antigens in this system that it has been estimated that one needs a panel of at least 2000 HL-A typed donors unrelated to the patient in order to expect to find one with a pattern similar to the patient's. The chance of compatability is much higher if one can use related individuals in the patient's own family, but those who believe marrow transplantation will be a successful form of therapy warn against using related individuals as blood donors. If one did, the patient might become sensitized against the individual who might otherwise be the best marrow donor for such a transplant.

If one is able to select platelet donors on the basis of HL-A compatabiltity, then there must be ways of harvesting four to six units of platelet concentrate from him at one time in order to provide satisfactory numbers of platelets for transfusion. Machines, essentially preparatory centrifuges, are available for platelet collections in these quantities, but such machines are expensive and not available in many blood banks. Trained personnel are required to operate these machines, and the donor must remain hooked up to it for at least one and one-half hours for the procedure. These requirements present definite logistic and motivational problems, especially if unrelated people are used as donors, but we anticipate that these services will be developed, at least at the larger community and regional blood bank centers.

Experimental evidence indicates that it is possible to freeze, store, and subsequently recover intact a significant number of platelets. Dimethyl sulfoxide (DMSO) is the cryoprotective chemical which has given the best results and hopefully, technics using glycerol, a very effective and safe agent for protecting red cells in frozen storage, can be extended to platelets. The best freezing technics recover only about thirty per cent of the platelets for transfusion. This does not compare favorably to the greater than fifty per cent recovery which is achieved with liquid storage, but it does provide a means by which an individual can store his own platelets for future use. Such autologous storage and therapy can be useful in leukemic pa-

tients and in others undergoing radiation or chemotherapy for cancer. Platelets drawn and frozen during periods of remission or before cancer therapy could be available for later effective use when leukemic relapse or cancer therapy toxicity produces thrombocytopenia.

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The clot: *Panel Discussion*

Robert Chapman, MD, John Franks, MD, Roger Hamstra, MD, John Kurnick, MD, and Stephen Wallner, MD, Editor

Query: Is there a treatment for the Antithrombin III deficiency state?

Dr. Wallner: Yes, since heparin is a co-factor for Antithrombin III action, we believe that these patients should receive heparin.

Query: Can Antithrombin III levels be used as a screening test in patients who are on birth-control pills?

Dr. Wallner: Yes, although very good clinical correlates are lacking, and the results of a single test may be misleading. If the Antithrombin III Level is in the range of those with the hereditary deficiency state who clot, I would stop the pills. Incidentally, the *in vitro* evidence of hypercoagulability reverts to normal in a matter of days when the estrogen is stopped.

Query: What is the usefulness of the immunologic reactive Factor VIII and IX?

Dr. Hamstra: From a diagnostic standpoint, it distinguishes well between patients with von Willebrand's disease and classical Factor VIII deficiency. It does not distinguish better than previously, when we made the diagnoses of von Willebrand's on the basis of moderately depressed Factor VIII levels plus the bleeding tendency and platelet functional alterations. Our diagnostic acumen is not better, but is confirmed by having this additional test. We have found that the immunologic determination of Factor VIII is particularly useful when evaluating the relatives of patients with Factor VIII deficiency or von Willebrand's disease. Our hemophilia treatment center here operates under a law passed by the Colorado legislature, which indicates our obligation to the entire state to give genetic counseling and to study the families of patients with hereditary bleeding disorders. Deficiency of Factors VIII and IX, and von Willebrand's represent the majority of our patients and we have been searching family trees to detect carriers. Identification by means of Factor VIII activity is not satisfactory. However, the precision of detecting carriers by plotting the immunologic Factor VIII level against the Factor VIII activity has a ninety per cent reliability in separating away the carriers of Factor VIII deficiency from those who are not carriers. I would not use this test right off, since it is sufficiently

difficult to get. If you have a question about von Willebrand's vs Factor VIII Deficiency vs no problem, I think the standard methods of assay are generally satisfactory.

Query: How is the serial thrombin time used?

Dr. Franks: The serial thrombin time is an assay which tells us something about the anticoagulant activity of the fragments that have been produced by hydrolysis or by lysis (plasma lysis) of the fibrinogen and fibrin.

Query: It was once stated that steroids should be tried for a minimum of three to six months before undertaking splenectomy in the patient with ITP. Has the feeling on that changed?

Dr. Kurnick: There are many possible answers to that question. Some hematologists favor splenectomy without any prior steroid treatment, while those of Dr. Dameshek's persuasion try to hold off as long as possible. There is usually a steroid response within three to four weeks, if one is going to occur, and I would give the patient about four weeks to respond unless he starts hemorrhaging massively, in which case an emergency splenectomy would be indicated. If the patient intitially responds and then drops his platelet count while on steroids, I would opt for a splenectomy, rather than wait to see if the count would rise again. One does not start tapering the steroids until the platelet count stabilizes, after which one reduces the doses gingerly in an attempt to get the patient off the drug completely. If the patient starts dropping his count during the tapering, I would increase the steroids to try to normalize the count again, and then go to the splenectomy. It may take three months to do all this in the initially responsive patient who relapses when the steroids are tapered.

Query: If a patient starts with a very low platelet count, say 10,000, is put on a good dose of steroids, say 60 mg/day, and no change occurs in two weeks, would you persist another two weeks in the face of that low a count?

Dr. Kurnick: If the patient is stable at 10,000 and clinically appears to be tolerating this level, I would wait for a total of, at most, four weeks, but I would not argue with someone who wanted to do the splenectomy sooner.

Dr. Hamstra: I would remove the spleen after two weeks if the count were stable.

Query: Are the hazards of the hypercoagulable state just as prevalent in menopausal women on estrogens as in younger women on the pill? If so, is there any rationale for using the Antithrombin III test then?

Dr. Hamstra: I do not know of any specific populations of older women on estrogens where hypercoagulability has been measured. I would think that it would occur regardless of age.

Dr. Kurnick: There is one study which showed that Antithrombin III lowering is proportional to the dose of estrogen in birth-control pills. (Conrad et al, *Lancet II,* 1148, 1972) and another which correlates the degree of Antithrombin III lowering on the pill with frequency of thrombotic problems Zuck et al, *Surg Gynecol Obstet* 133:609, 1971) Thus it might well be worthwhile to test Antithrombin III levels in Postmenopausal women on estrogen.

Query: Dr. Wallner has indicated that individuals with Antithrombin III depression were treated with heparin. It is very difficult to treat an out-patient with heparin. Is there a better mode? Please discuss treatment with coumadin.

Dr. Hamstra: I do not think the coumadins are worth very much in this situation. Our experience with these people is that they continue to clot and have recurrent thrombophlebitis when they are managed on coumadin. If you really drive hard with the drug, and keep the prothrombin time in the range of 2-1/2 times control, so that the patients will bleed freely from little nicks and ooze blood from their gums when brushing, you might get by. But this is an uncomfortable level. You are running them at a 15 to 10 per cent prothrombin time on a per cent scale. If you let them get up to 20 to 25 per

cent, phlebitis may occur. We have tried coumadin in some of these patients after using heparin for a few years, not because the heparin failed, but because the patient developed osteoporosis, secondary to the long-term heparinization. The coumadin drug is an inferior agent compared to heparin for managing the problem, but it has something to offer when you push hard, but then you risk complications from coumadin to keep the disease under control.

Query: Dr. Franks, what is your recommendation for a preventive drug to give patients with recurrent phlebitis?

Dr. Franks: I think I would have to go along with what has just been said and use heparin.

Query: What about the platelet inhibitors?

Dr. Franks: We have not tried them and most of the work out of Seattle indicates that platelet inhibitors may work for problems in which platelet clots are forming, i.e., on the arterial side, but that they are not useful in the ordinary variety of recurrent thrombophlebitis.

Query: At one time Dr. von Kaulla was interested in the possibiltity that niacin or nicotinic acid might activate some endogenous or internal thrombolytic system. Can you comment on his more recent thinking in this regard?

Dr. Franks: Dr. von Kaulla and I discussed the use of nicotinic acid derivatives as a means of handling intravascular coagulation. It is reported that these substances have a profound effect on labeled fibrinogen in the circulation. We do not think that the nicotinic acid derivatives are the final answer in activating the fibrinolytic system.

Query: What are your feelings about the practice here on the Orthopedic Service concerning the mini-dose of heparin for hip fracture therapy?

Dr. Hamstra: At Fitzsimons Hospital in excess of 200 cases have been managed with low-dose heparin therapy and their surgeons are impressed positively that the rates of pulmonary embolism and phlebitis are much reduced. They are also impressed that their patients bleed more, but they are ready to accept this for the decrease in clotting complications.

Dr. Franks: I just had sent to me and analyzed, some data from fifty-two cases which were studied by Dr. J. W. L. Davies, in Birmingham. These were studies of patients who had fractures of the hip. Many of these were elderly. Dr. Davies did essentially what we are doing here. He studied radioactive fibrinogen metabolism in these people. He found clear-cut evidence of a chronic intravascular process of coagulation and retention of fibrin in and around the wound. This process could be reversed by heparin.

Query: What specific determinant do you use to control your heparin doses?

Dr. Franks: I use the activated clotting time (ACT test).

Dr. Hamstra: One can use a whole blood-clotting time, or a partial thromboplastin time, but the most sensitive way is to use a thrombin time. It is a little tricky to run in the laboratory. We have sort of compromised here. In this institution almost all patients on the medical service, a good number of patients on the surgery service, and virtually all renal dialysis patients are being managed by a test called the Harem test (the Heparin Assay Rapid Evaluation Method). It is a whole-blood, activated, partial thromboplastin time that can be done right at the bedside by our housestaff team. Our housestaff are happy with it, or the ACT test, because it gives them an answer quickly.

Query: Can you over-heparinize a patient to where he is at more risk of bleeding?

Dr. Hamstra: We have all been involved in cases in which an error was made in which an excessive dose of heparin was given to a patient, after which he had an infinite clotting time for days and yet did fine. However, until more studies have been done, we feel it is prudent to maintain control over heparinization.

Employability of cancer patients

M. B. Bond, MD*, Basking Ridge, N. J.

Cancer may threaten the ability to work and thus to earning a living. Frequency and severity rates for cancer in a large employed group are presented and discussed.

Cancer is a serious yet common disease in the United States as well as the world. It is most common in middle-aged and elderly persons, although it is the second most frequent cause of death during the low death-rate years of childhood. Thus this serious disease strikes persons often during their most active and productive years, and it is therefore a serious social and economic problem as well as a threat to life.

Diagnosis of cancer may indeed be grave news for an individual and his family. One of many concerns is the outlook for continued employment. Here the news is not always so bad. This report covers the occurrence of cancer and the employment status in a population of 763,256 employees in Bell System telephone companies throughout the United States for a twelve-month period, July 1974 through June 1975.

Method

All employees with more than six months service are eligible to receive benefits for disability occurrences of more than seven days. Attending physicians provide certificates to our medical departments stating the cause of the disability in each case. Such employees are also usually seen by our own physicians during long disability periods or upon return to work. Information about each terminated case of disability is coded, and transmitted to my office for processing.

Disability payments are continued until return to work or for a maximum of one year. Employees whose disease or treatment results in impairments that prevent return to their regular jobs are placed on other jobs for which they are suited. If disability persists more than a year, employees are given pensions if they have more than fifteen years service. Their health care insurance is continued.

Data on individual cases are processed to produce frequency and severity rates. Frequency is the number of cases per 1000 active employees; severity is the average days' disability per case. Cause specific rates by age and sex for the more common sites are calculated. Since an individual might have two cases of cancer disability of more than seven days in one year, the number of cases exceeds slightly the number of persons.

TABLE 1

Characteristics of Populations at Risk, 1974-75

	Male	Female
Number	377,231	386,025
Mean age	37.7	34.8
Per Cent	49.4%	50.6%
Frequency rate	1.64/1000	2.57/1000

Results

For the twelve month period there were 1697 cases of disability among 1613 persons for a frequency rate of 2.11 persons with cancer per 1000 employees. Of the 763,256 employees, 386,025 (50.6 per cent) were females and 377,231 (49.4 per cent) were males. The unadjusted frequency rate in females was 2.57 (993 persons) as compared to the male rate of 1.64 (620 persons).

Table 2 shows the numbers and frequency rates for total cancers plus those of more common anatomical sites in males. Nearly all the respiratory cancers were of the lung and bronchus. Gastrointestinal and genitourinary cancers were almost as common as respiratory. Also shown in Table 2 are followup data during the one year period. An indication of the relative seriousness of the different sites can be seen in the numbers who died during the study year and the numbers who returned to work. This is also a measure of the rapidity of growth and metastases, as well as of the effectiveness of therapy. Lung cancer is not only most common but also most deadly.

Similar data for females are shown in Table 3. Table 4 shows the age and sex specific frequency rates for all malignancies. These figures are similar to data reported by Stone on the Bell System employees for 1970-71.[1] Cancer occurs usually in persons beyond the age of 40, with 82 per cent in males and 77 per cent in females occuring in this age group.

* Corporate Medical Director, American Telephone & Telegraph Company, Basking Ridge, New Jersey. Presented at The Rocky Mountain Cancer Conference, Denver, July 16, 1976.

TABLE 2

Cancer Morbidity and Mortality
Male Employees
1974-75

Males	Totals	Frequency	Death In Study Year	Returned To Work	Benefits Expired +
All cancers					
*ICDA 140-209	620 (100.0%)	1.64	145 (23.4%)	438 (70.6%)	37 (6.0%)
Gastrointestinal ICDA 150-159	103 (100.0%)	.27	31 (30.1%)	64 (62.1)	8 (7.8%)
Respiratory ICDA 160-163	109 (100.0%)	.29	48 (44.0%)	52 (47.7%)	9 (8.3%)
Genitourinary ICDA 185-189	103 (100.0%)	.27	11 (10.7%)	88 (85.4%)	4 (3.9%)
All other cancers	305 (100.0%)	.81	55 (18.0%)	234 (76.7%)	16 (5.2%)

+ After disability benefits of one year.
* International Classification of Diseases, Adapted, Eighth Revision

Discussion

Cancer is diagnosed much more frequently in females than in males among our employees. However, gastrointestinal cancer is slightly more common in males and respiratory cancer is three times as common among our males. Genitourinary cancers are two and a half times more frequent in females, indicating these are genital rather than cancers of the similar urinary organs. Respiratory cancers are more deadly in females, with more than half dying during the study year as compared to slightly less than half in the males. Chronicity and/or effectiveness of therapy can be assumed in breast and female genitourinary cancers from the small numbers who died during the year and the fact that more than 85 per cent returned to work. These two classes of cancer make up more than half the female cancers. Except for breast and genitourinary cancers, the frequency rates are higher in males for comparable sites as well as "all other cancers."

The average disability or severity was 93.3 days in males and 108.3 days in females. In comparison with other causes of disability, cancer patients have the greatest severity, exceeding that of diseases of the circulatory system (males) or mental disorders (females) by about one-third. However in terms of frequency, cancer ranks only 14th in males and 15th in females in the Bell System. In total disability days, counting only those cases lasting more than a week, cancer cases constitute only 3.9 per cent. Length of disability is not necessarily a good indication of seriousness of disease in our population, whether it be cancer or other illness. Return to work is perhaps a better overall indicator of seriousness, and our data show that 70.6 per cent of males and 78.8 per cent of females return to work after a disability due to cancer. Similarly, 23.4 per cent of males died from their cancer during the study year whereas only 13.7 per cent of females died. Six per cent of the males and 7.45 per cent of the females remained off work for a full year. Nearly all of these employees had fifteen or more years service and were placed on pensions.

TABLE 3

Cancer Morbidity and Mortality
Female Employees
1974-75

Female	Totals	Frequency	Death in Study Year	Returned To Work	Benefits Expired +
All cancers					
*ICDA 140-209	993 (100.0%)	2.57	136 (13.7%)	783 (78.8%)	74 (7.45%)
Gastrointestinal ICDA 150-159	80 (100.0%)	.21	23 (28.8%)	49 (61.3%)	8 (10.0%)
Respiratory ICDA 160-163	43 (100.0%)	.11	25 (58.1%)	16 (37.2%)	2 (4.7%)
Breast ICDA 174	363 (100.0%)	.94	26 (7.2%)	311 (85.7%)	26 (7.2%)
Genitourinary ICDA 180-184	243 (100.0%)	.63	23 (9.5%)	209 (86.0%)	11 (4.5%)
All other cancers	264 (100.0%)	.68	39 (14.8%)	198 (75.0%)	27 (10.2%)

+ After disability benefits of one year.
* International Classification of Diseases, Adapted, Eighth Revision

These data have not considered the problem of the individual who has had cancer but is an applicant for employment rather than already employed. We do not automatically reject such persons although prior to a few years ago this practice was not uncommon with many employers. We consider each such applicant on an individual basis, and if the cancer has been adequately treated and the individual is able to work on a regular basis, then we consider him or her for hire on the basis of ability to do the job.

During the past few years our practices of hiring cancer patients and others with a history or presence of serious illness have become consistent throughout our many companies. Our practices are in conformity with Regulations of the Federal Department of Labor, implementing the Rehabilitation Act of 1973, commonly called the Handicapped Law. It is against the law to refuse to hire a person with a history of, or the presence of, cancer unless it can be shown the individual cannot do the job. Many states have such laws now, some of which are more stringent than the federal regulations.

Cunnick et al[2] have reported their experiences in the Metropolitan Life Insurance Company in hiring persons with a history of cancer. In their experience, applicants with a cancer history are rare, with fewer than one per thousand applicants. Of these, about half were found suitable for hire after evaluation. And of those hired most had good employment records. Such persons are not an unusual burden on the insurance benefits given to all employees, Cunnick concluded.

I have not seen other reports on experiences with the hiring of persons with a past or present history of malignant disease. Cancer occurs mostly in mid-life and beyond, when persons are usually established in their work, and therefore they do not often appear in the labor market seeking jobs, at least in Cunnick's experience. His report covered only clerical and sales jobs and the same may not be true of all classes of workers. At any rate, the recent federal regulations on handicapped

TABLE 4

Cancer Age-Specific Rates
1974-75

Age	Cases per 1,000 Male	Female
Under 20	.3	.3
20 - 29	.5	.7
30 - 39	.7	1.8
40 - 49	1.9	4.1
50 - 59	4.9	7.6
60	9.9	9.0
Percent of cases 40 years and older	82%	77%

as well as the many state laws on employment should assist treated cancer patients in getting jobs.

Conclusions

Cancer was diagnosed in Bell System employees at a frequency rate of 2.11 cases per 1000 employees during a one year period. The rate for females was 2.57 and for males 1.64. Cancer in males ranks 14th and in females 15th in frequency of disability cases lasting more than a week. However the average length of disability (severity) in cancer cases is 93.3 days (males) and 108.3 days (females) which is greater by a third than that of any other cause.

Most employees with a disabiltity due to cancer return to work — 70.6 per cent of males and 78.8 per cent of females. However 23.4 per cent of males died during the study year as did 13.7 per cent of females.

The lung and bronchus was the most frequent site of cancer in males and this type is the most deadly with nearly half dying during the study year. Breast was the most common site of cancer in females, with genitourinary cases next most frequent. More than 85 per cent of such employees returned to work, indicating slow progression and/or effectiveness of therapy.

For the cancer patients who are able to work we have no problems in continuing them at work. The new federal regulations on handicapped should prevent unemployed persons with a history of cancer from having difficulty in finding jobs.

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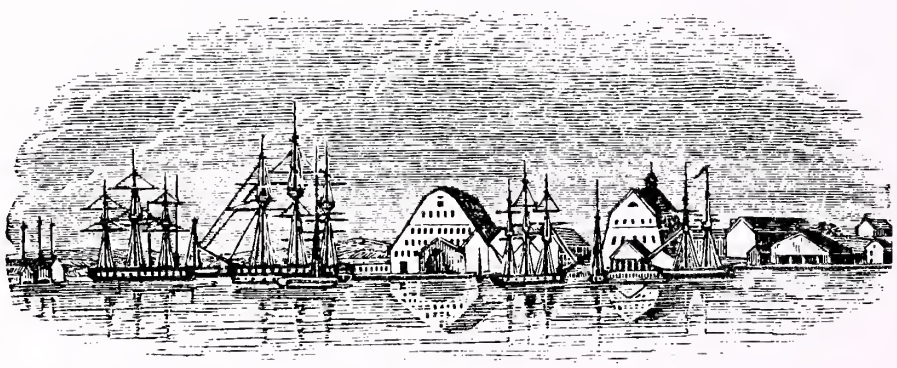

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Saturday, June 18, 7:30 AM–Noon
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SF-13. Basic Life Support—Cardiopulmonary Resuscitation (CPR)

Saturday, June 18, 1:00-5:30 PM
(5-hour course: $50)

SF-14. Basic Life Support—Cardiopulmonary Resuscitation (CPR)

Saturday, June 18, 9:00 AM–Noon–1:00–5:00 PM &
Sunday, June 19, 9:00 AM-Noon
(11-hour, 2-day course: $130)

SF-15. Public Speaking Seminar

Saturday, June 18, 8:00-11:00 AM
(3-hour course: $30)

SF-16. Interpretation of Chest Roentgenograms

Saturday, June 18, 2:00-5:00 PM
(3-hour course: $30)

SF-17. Clinical Electromyography for the Referring Physician

Sunday, June 19, 8:00–11:00 AM–2:00–5:00 PM
(6-hour, 1-day courses: $60)

SF-18. Electrocardiography: Basic Concepts
SF-19. Diagnosis & Management of Cardiac Arrhythmias
SF-47. Dermatology for Nondermatologists
SF-21. Antibiotic Usage in Pediatrics
SF-22. Primary Treatment of Injuries of the Hand
SF-23. Gynecologic Endocrinology
SF-24. Exercise in the Diagnosis, Prevention, & Rehabilitation of Ischemic Heart Disease
SF-25. Therapy of Sexual Dysfunctions & Disorders
SF-26. Current Bedside Clinics in Neurology

Sunday, June 19, 7:30 AM–Noon–1:00–5:30 PM &
Monday, June 20, 7:30 AM-Noon
(12-hour, 2-day course: $120)

SF-27. Advanced Life Support—Cardiopulmonary Resuscitation (CPR), Prerequisite: Basic Life Support course; *Required Reading: CPR Advanced Life Support Text* 70034B, $7.50; order through American Heart Association, 7320 Greenville Ave., Dallas, Tex. 75231—make check payable to: American Heart Association

Sunday, June 19, 8:00-11:00 AM
(3-hour courses: $30)

SF-28. Liver Disease—New Developments
SF-29. Useful Diagnostic Laboratory Tests in Endocrine Disease
SF-32. Refresher Course in Dermatopathology
SF-70. Diagnosis & Treatment of Fractures of the Lower Extremities

Sunday, June 19, 2:00-5:00 PM
(3-hour course: $30)

SF-20. Live Case Presentations for Dermatologists
SF-30. Useful Diagnostic Laboratory Tests in Endocrine Disease

Monday, June 20, 8:00–11:00 AM–2:00–5:00 PM & Tuesday, June 21, 8:00–11:00 AM–2:00 to 5:00 PM (12-hour, 2-day course: $120)

SF-31. Practical Exercises in Emergency Care

Monday, June 20, 8:00–11:00 AM–2:00–5:00 PM (6-hour, 1-day courses: $60)

SF-33. How to Sew (Principles of Suture Technique)
SF-34. Problem Care Seminar on Common Hand Injuries: Basic & Advanced Techniques
SF-35. Respiratory Emergencies in Children
SF-36. Critical Injuries: Recognition & Early Treatment
SF-37. Evaluation & Management of Respiratory Failure
SF-38. Spine & Spinal Cord Injuries: An Update for Primary Care Physicians
SF-39. Peptic Ulcer
SF-40. Diabetes
SF-41. Practice Management Seminar
SF-42. An Overview of Current Psychotropic Drugs

Monday, June 20, 8:00–11:00 AM (3-hour course: $30)

SF-44. Management of Convulsive Disorders: "Fits Are Old—Not So Appraisal or Treatment"
SF-71. Diagnosis & Treatment of Fractures, Dislocations, & Epiphyseal Injuries in Children

Monday, June 20, 1:00-5:30 PM (5-hour course: $50)

SF-43. Basic Life Support—Cardiopulmonary Resuscitation (CPR)

Tuesday, June 21, 8:00–11:00 AM–2:00–5:00 PM (6-hour, 1-day courses: $60)

SF-45. Acid-Base, Fluids, & Electrolytes
SF-46. Echocardiography: Basic Concepts
SF-48. Some Things Old & Some Things New in the Treatment of Rheumatic Diseases
SF-49. New Drugs in Gastroenterology
SF-50. Urologic Pathology
SF-52. Hypertension 1977: A Practical Approach to the Evaluation & Management
SF-53. Common Errors in Emergency Wound Management: Basic & Advanced Techniques
SF-54. Complete Management of Pulmonary Neoplasms
SF-55. Office & Hospital Management of Anorectal Disease

Tuesday, June 21, 7:30 AM–Noon–1:00–5:30 PM & Wednesday, June 22, 7:30 AM-Noon (12-hour, 2-day course: $120)

SF-56. Advanced Life Support—Cardiopulmonary Resuscitation (CPR), Prerequisite: Basic Life Support course; see SF-27 for required reading

Tuesday, June 21, 8:00–11:00 AM (3-hour course: $30)

SF-51. CT Scanning & Neurological Diagnosis: "New Views of the Old Head"
SF-72. Diagnosis & Treatment of Fractures of the Upper Extremities

Tuesday, June 21, 2:00–5:00 PM (3-hour courses: $30)

SF-57. Biofeedback in Muscle Re-Education & Relaxation
SF-58. Toward More Appropriate Utilization of Laboratory Tests: Maximizing Clinical Information from Laboratory Tests

Wednesday, June 22, 8:00 AM–Noon–2:00–5:00 PM (6-hour, 1-day courses: $60)

SF-59. Electrocardiography: Basic Concepts
SF-60. Diagnosis & Management of Cardiac Arrhythmias
SF-61. Pediatric Dermatology
SF-62. Recent Advances in GI Surgery
SF-64. Diagnosis & Therapy of Common Renal Diseases
SF-65. Office Orthopedics for the Nonorthopedist
SF-66. Drug Interactions Relevant to the Primary Care Physician
SF-67. Practical Office & Laboratory Procedures in Allergic Disease

Wednesday, June 22, 8:00–11:00 AM (3-hour course: $30)

SF-63. On Understanding & Treating Dizziness
SF-69. Office Urology

Wednesday, June 22, 1:00–5:30 PM (5-hour course: $50)

SF-68. Basic Life Support—Cardiopulmonary Resuscitation (CPR)

Registration Form

AMA's 126th Annual Convention/San Francisco/ June 18-22, 1977

Please print and return to:

AMA Dept. of Meeting Services, 535 N. Dearborn St., Chicago, IL 60610; or phone: (312) 751-6503.

I am a member of the AMA through the following state medical association: _____. If you are not an AMA member, please add $35 to your total remittance. ($35 fee does not apply to medical students or residents.) Payment must accompany choice of courses (where fees are required) on this registration form.

Advance Registration. To receive your Advance Registration Card, this form must arrive at the AMA by May 20, 1977. (After this date, please register at Brooks Hall CME Registration Desk, San Francisco, California. Your Advance Registration Card will be sent to you on May 27, 1977, unless you request an earlier mailing date.

General Registration. AMA members and their guests, no fee. Nonmember physicians, $35; their guests, $10 per person. Medical students, residents, and Canadian and other foreign physicians, no fee. (Does not include CME course fees.)

CME Course Registration. Please Note: Fee schedule for each item. Insert course numbers of your choice for each day and indicate your order of preference in the space provided. If minimum registration is not attained for your first choice, your alternate choice will be substituted. The course numbers correspond to the listing and fees shown.

All medical students and residents are entitled to a 50% discount on course registration fees ($10 per credit hour is standard rate). Payment must accompany choice of courses requested on this registration form.

Date		Course No.	Fee	Course No.
Sat. June 18	AM	1st Choice_____	$____	2nd Choice_____
	PM	1st Choice_____	$____	2nd Choice_____
Sun. June 19	AM	1st Choice_____	$____	2nd Choice_____
	PM	1st Choice_____	$____	2nd Choice_____
Mon. June 20	AM	1st Choice_____	$____	2nd Choice_____
	PM	1st Choice_____	$____	2nd Choice_____
Tues. June 21	AM	1st Choice_____	$____	2nd Choice_____
	PM	1st Choice_____	$____	2nd Choice_____
Wed. June 22	AM	1st Choice_____	$____	2nd Choice_____
	PM	1st Choice_____	$____	2nd Choice_____
		Add $35 if Non-Member	$____	
		Total	$____	

Please print or type:

Name _____

Office Address _____

City/State/Zip _____

Office Phone No. ( ) _____

SEE REVERSE SIDE FOR HOUSING INFORMATION BEFORE COMPLETING FORM BELOW

Housing Form

American Medical Association's Annual Convention
AMA Housing Bureau c/o San Francisco Convention & Visitors Bureau
1390 Market Street Suite 260
San Francisco, California 94102

Please print or type six choices:

1. _____
2. _____
3. _____
4. _____
5. _____
6. _____

Please enter my reservation at the above hotel/motel for:

Single(s)	**Double(s)**	**Twin(s)**
_____at $_____	_____at $_____	_____at $_____

Suite(s)

_____at $_____ 1 Bedroom

_____at $_____ 2 Bedroom

CANCELLATIONS—Please notify HOUSING BUREAU, 1390 Market Street, Suite 260, San Francisco, California 94102 of all CANCELLATIONS UP TO 15 DAYS PRIOR to Convention. WITHIN LAST 15 DAYS MAKE CANCELLATIONS DIRECT WITH HOTEL.

CHANGES—ALL OTHER CHANGES TO BE MADE DIRECTLY WITH HOTEL AT ALL TIMES.

Rates subject to local tax

CONFIRMATIONS WILL BE MAILED UP TO JUNE 6, 1977

Room will be occupied by:

Name_____
(Please print or type)

Firm_____

Address_____

City/State/Zip Code_____

Office Phone Number (_____)_____-_____

Additional Occupants (List ages of children, if any):

_____

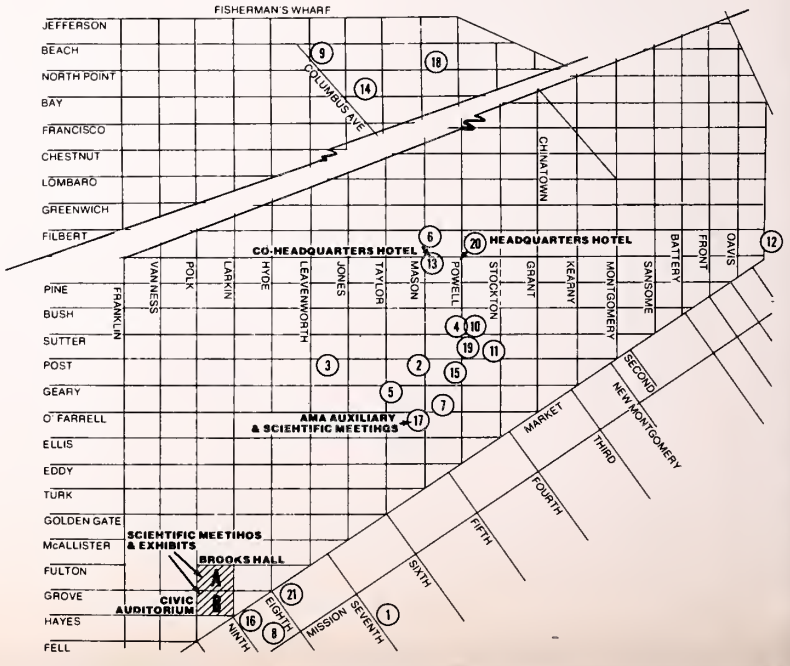
Arrival Date _____ at _____ AM; PM;

Departure Date _____

126th AMA Annual Convention
June 18-22, 1977
San Francisco, California

AMA HOUSING BUREAU c/o
San Francisco Convention & Visitors Bureau
1390 Market Street • Suite 260
San Francisco, California 94102

Key # HOTEL/MOTEL	Single	Double	Twin	Suite
1. Americania Motor Lodge	$27	$31	$35	$42 (1 Bedroom)
2. Barrett Motor Hotel	$38	$44	$44	$92 (1 Bedroom) $136 (2 Bedroom)
3. Bedford Hotel	$19	$24	$26	$45 (1 Bedroom)
4. Cartwright Hotel	—	$23	$24	—
5. Clift Hotel	—	$60	$60	$150 (1 Bedroom)
6. Fairmont Hotel	Headquarters Hotel • House of Delegates			
7. Handlery Motor Inn	$34-$36	$39-$43	$39-$43	—
8. Holiday Inn-Civic Center	$29	$35	—	$65-$73 (1 Bedroom) $87-$92 (2 Bedroom)
9. Holiday Inn-Fisherman's Wharf	$39-$42	$45-$50	$45-$50	$60 (1 Bedroom)
10. Holiday Inn-Union Square	$42-$45	$48-$51	$48-$51	$81 (1 Bedroom) $132-$162 (2 Bedroom)
11. Hyatt on Union Square	$38-$60	$53-$75	$53-$75	$125-$175 (1 Bedroom) $300-$350 (2 Bedroom)
12. Hyatt Regency San Francisco	$34-$58	$44-$68	$44-$68	$85-$175 (1 Bedroom) $250 (2 Bedroom)
13. Mark Hopkins Hotel	Co-Headquarters • House of Delegates			
14. Ramada Inn-Fisherman's Wharf	$38	—	$46	$80 (1 Bedroom)
15. St. Francis Hotel	$35-$65	$45-$80	$45-$80	available upon request
16. San Franciscan Hotel	$30-$34	$34-$38	$34-$38	$65-$85 (1 Bedroom)
17. San Francisco Hilton	$32-$62	$43-$73	$43-$73	$107-$135 & up (1 Bedroom) $190-$200 & up (2 Bedroom)
18. Sheraton at Fisherman's Wharf	$37-$45	$45-$53	$44-$52	$100 (1 Bedroom) $200 (2 Bedroom)
19. Sir Francis Drake	$32-$54	$40-$60	$40-$60	$145 (1 Bedroom) $185 (2 Bedroom)
20. Stanford Court	Co-Headquarters Hotel • House of Delegates			
21. TowneHouse Hotel	$28-$34	$30-$35	$30-$36	$64 (1 Bedroom) $95 (2 Bedroom)



Nature just doesn't make a perfect food.

No one food supplies all the nutrients children need.

But milk supplies more essential nutrients per calorie than most other foods.

A child between the ages of six months and six years, for example, can get at least three-fourths of his daily dietary allowance for calcium, riboflavin, vitamins D and B_{12}, phosphorous, and protein from just three glasses of milk. And milk is also a good source of vitamins A and B_6, as well as thiamin and niacin (please see chart).

That's why the U.S.D.A., acting on the recommendation of the National Research Council—National Academy of Sciences, has established these guidelines for milk consumption:

Children up to 6 years
Two to three 8 oz. glasses per day.

Children 6 to 12 years
Three or more 8 oz. glasses per day.

For a free copy of the Dairy Council brochure "Food Before Six," contact Dairy Council, 12450 N. Washington, Thornton, CO 80241.

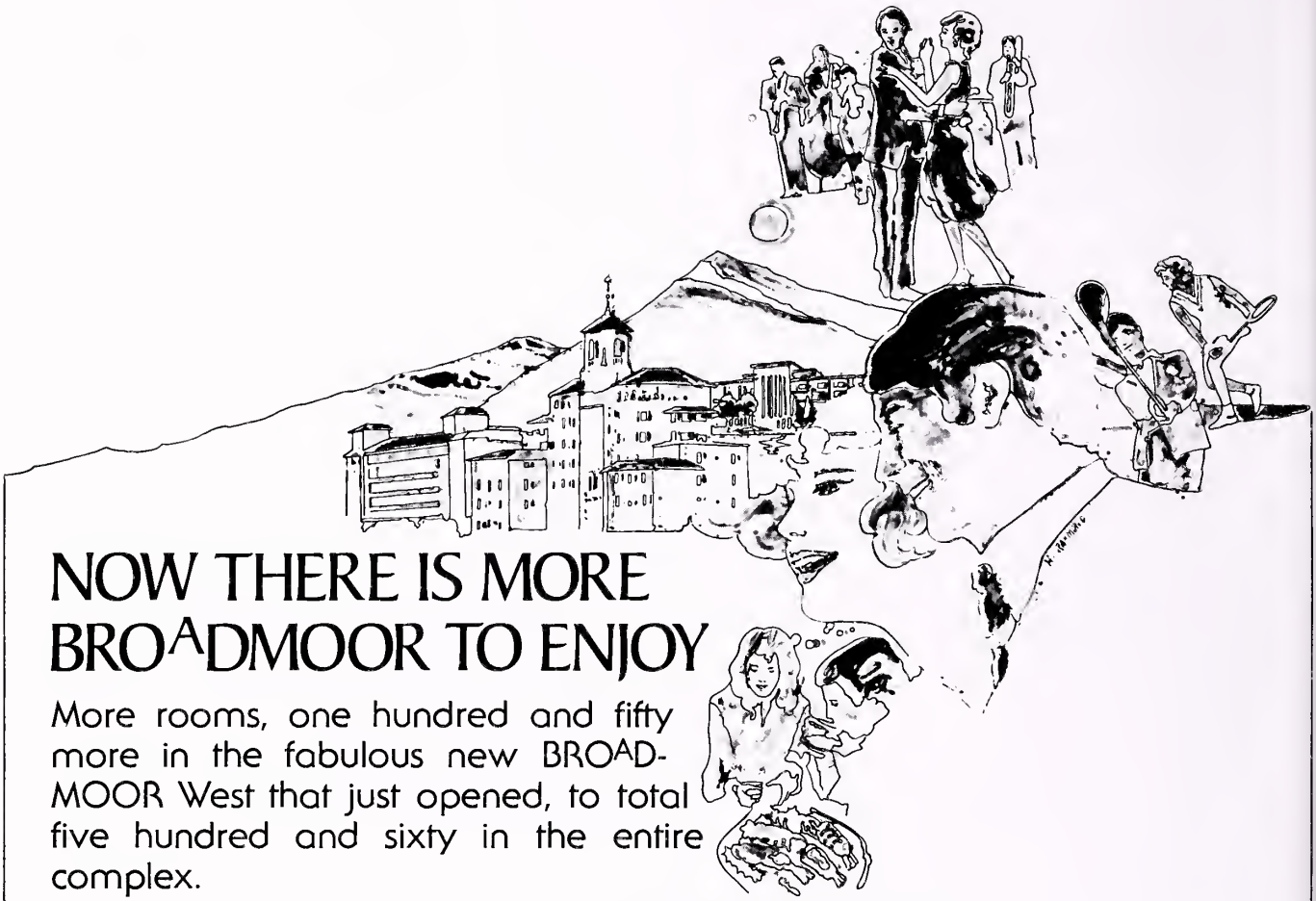
Per cent of Recommended Daily Allowance contributed by three 8 oz. glasses of fortified milk.

	6 mos.—1 yr.	1 yr.—3 yrs.	3 yrs.—6 yrs.
Calcium	100%	100%	100%
Riboflavin	100	100	100
Vitamin B_{12}	100	100	100
Protein	100	100	80
Phosphorus	100	85	85
Vitamin B_6	76	51	34
Vitamin D	75	75	75
Niacin*	71	63	47
Thiamin	56	40	31
Vitamin A	46	46	37

*maximum niacin equivalents based on: 60 mg tryptophan=1 mg niacin

ndc **National Dairy Council**

Milk. Sometimes we forget all the good things it does.



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More meeting facilities, nearly double what we have provided. Over thirty conference rooms are now available, with never a charge for their use. They vary in size from a sixteen person capacity Directors Room, to two ballrooms, an exhibit hall, and the International Center that seats 1,600 for banquets and 2,400 for conferences.

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COLORADO

Kellogg Foundation Awards Support

A grant of $168,618 to Swedish Medical Center, Englewood, Colorado, will make possible a prototype life support program in Douglas County, Colorado, in a major grant from the Kellogg Foundation of Battle Creek, Michigan.

According to Robert A. DeVries, Kellogg Foundation program director, "the grant is for training critical care nurse practitioners, and purchasing medical telemetry equipment for the prototype Life Support Center being constructed in Douglas County near Castle Rock."

A county-wide fund-raising program for $100,000 for the center is soon to start. More than 50 Denver and Arapahoe County physicians will voluntarily assist the program which focuses on the nurse practitioner who is to receive extensive emergency and critical care training under the grant. Robert Brittain, MD, will be in charge of the training program.

The Life Support Center is to be staffed 24 hours a day by these highly trained nurse practitioners under close supervision of emergency physicians at Swedish via two-way communications and medical telemetry equipment.

Construction was started in February on a 20-acre site in Silver Heights north of Castle Rock. October completion is planned.

Colorado Internist Elected Secretary-Treasurer

John F. Farrington, MD, Boulder, was re-elected secretary-treasurer of the American Society of Internal Medicine at its annual meeting in March in San Francisco. Dr. Farrington served as president of the Colorado Society of Internal Medicine from 1970-71, and president of the Boulder County Medical Society from 1965-68. He is a former associate medical director of the Colorado Foundation for Medical Care, and chairman of its Health Standards Committee.

Ultrasound Developer To Be Honored

A banquet at the Marriott Hotel in Denver on May 25 will honor Joseph H. Holmes, MD, who will retire July 1 after 42 years of service in medicine as professor of medicine and radiology at the University of Colorado Medical Center. Holmes is one of the earliest developers of diagnostic ultrasound and an innovator in renal medicine. The Joseph H. Holmes Fund has been established to create the Joseph H. Holmes Visiting Professorship in Innovative Medicine.

NEW MEXICO

Albuquerque Doctor Presents Papers in East

Robert Kellner, MD, Chief of psychiatry service at the Veterans Administration hospital, Albuquerque, will present a paper on "Research Findings in Psychoneurosis" at the Veterans Administration Hospital at Lexington, Kentucky, and "The Education of the Medical Student in Thanatology" at Columbia University Medical Center, New York in April.

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OBITUARIES

Colorado

Doctor **William A. Merritt,** died Wednesday, March 16, 1977 in Walsenburg, Colorado, where he had been ill for several months.

He was a native of Huerfano County, having been born there in 1903. He attended Valparaiso University at Valparaiso, Indiana, then received his doctorate from the University of Colorado in 1931. After an internship served in St. Paul's Hospital, Dallas, Texas, he opened an office for general practice in Aguilar, Colorado in 1934.

Save for a few months spent in Glenwood Springs in 1955, he practiced nearly 40 years in Huerfano County, Colorado.

He was a Mason and a member of the Huerfano County and Colorado Medical societies and the American Medical Association. He was quite active in medical society affairs, and served a term as president of the Huerfano Memorial Hospital staff.

Two sons, Brian and Bruce, survive, as does a daughter, Lois Merritt of Aguilar.

Doctor **John A. Egan** died in Denver March 15, 1977. Born May 9, 1903 in Parsons, Kansas, he was educated in the public schools of Wabash, Indiana, and attended the University of Indiana from which he received his MD in 1928. After a year of internship at the Indianapolis City Hospital, he opened an office for general practice in Greencastle, Indiana. In 1932 he entered the military service, and received his training in ophthalmology and otolaryngology at Walter Reed General Hospital in Washington, D.C. He was certified by the American Board of Ophthalmology in 1941.

He left the military service in 1948, and established a practice in ophthalmology in Denver. He was a teaching assistant at the University of Colorado School of Medicine.

Doctor Egan was a member of the Denver and Colorado Medical societies, and of the American Medical Association. He belonged to the American Academy of Ophthalmology and Otolaryngology, and the Colorado Ophthalmological Society.

He was married to Helen Service in 1930, and they had one child, Sandra Sue Egan, and later were divorced. In 1973 he was married to Betty A. Albright of Denver. In addition to his widow, he is survived by his daughter, Mrs. Sandra Sue Rowe of Divide, Colorado, and a brother, Maurice, of St. Simons Island, Georgia.

Doctor **Thomas W. Moffatt** died March 18, 1977. Born in Dubuque, Iowa, January 17, 1915, he attended Loras College in that city, then received his MD from the University of Iowa in 1940. After an internship at St. Joseph Hospital, Denver he took graduate training in dermatology at Barnard Skin and Cancer Hospital, an affiliate of Washington University in St. Louis, Missouri. After an additional year of graduate training at the University of Colorado, he opened an office in Denver for the practice of dermatology.

He was certified by the American Board of Dermatology and Syphilology in 1953. He was a member of the Denver and Colorado Medical societies, and of the American Medical Association. He was very active in the affairs of the Denver and Colorado societies, serving on numerous councils and committees.

He held memberships in the Denver Dermatological Society, the Rocky Mountain Dermatological Society, the Pacific Dermatological Society, the American Academy of Dermatology, and the Rocky Mountain Academy of Industrial Medicine. He served as president of the Rocky Mountain Dermatological Society in 1957.

Doctor Moffatt was married on December 16, 1941 to Marie Elizabeth Scartezina, and she survives, as do three sons, Thomas W. Moffat, Jr., MD, of Denver; Robert W. and James L. of Lakewood, Colorado, and a daughter, Miss Shauna Marie Moffatt of San Diego, California.

OVERSEAS EMPLOYMENT OPPORTUNITIES

Kwajalein, Marshall Islands

PHYSICIANS

Two full-time positions available now for family doctors experienced in general practice and/or pediatrics. One position also available for a general surgeon. The staff of physicians, together with a total hospital staff of about 50 persons, provide both inpatient and outpatient medical services for an island community of about 3500 persons, both civilian and military, including families with children, and single status men and women.

The 28-bed Kwajalein Hospital is modern and recently enlarged, and has a fully equipped outpatient clinic to handle the large flow of outpatients, plus excellent laboratory facilities.

Tropical climate on a lovely coral atoll. Fantastic scuba and snorkeling, tennis and golf.

Modern family housing available. Excellent, fully-accredited schools, kindergarten through high school.

U.S. Citizenship required. Comfortable living conditions. One year contracts. (Some shorter assignment of 3 to 6 months sometimes available on a single status basis). Vacation, bonus and other fringes. Potential U.S. Federal tax exemption. Reasonable cost of living.

Interested persons should RUSH curriculum vitae to: Clyde E. Albert, P.O. Box 12156, Oakland, Calif. 94604. An equal opportunity affirmative action employer.

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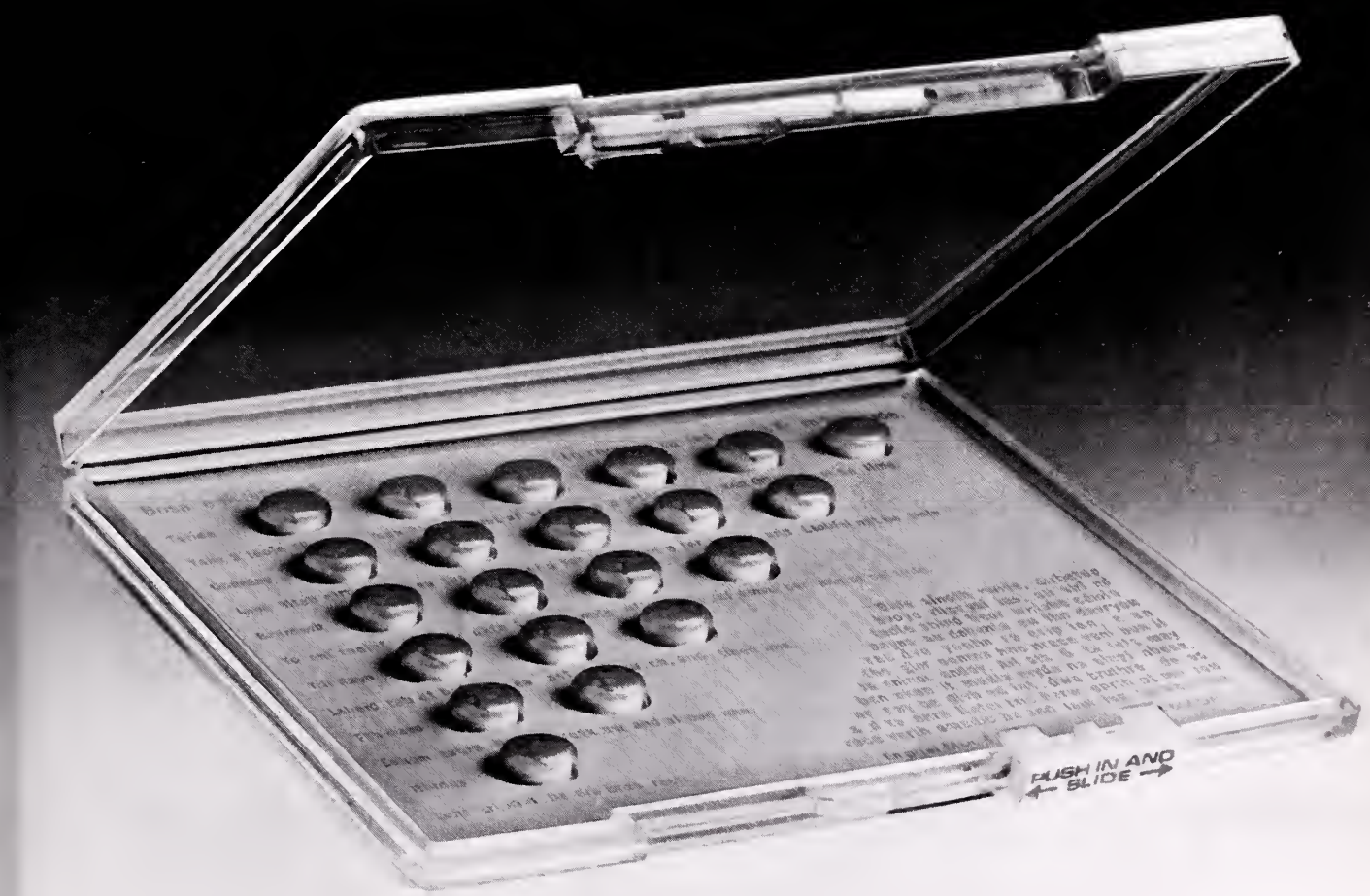

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MEDICAL BOOKS

The University of Colorado Medical Center Bookstore is proud to serve the medical community of the Rocky Mountain Area. Our stock of over 20,000 medical titles makes us the largest medical book merchandiser in this region. We offer our clientele a current and comprehensive selection of scientific texts and references, a special order department, and an easily accessible location.

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Recent publications now in stock include:

Billups - *American Drug Index*. Lippincott, 1977. ($14.50)
Hart - *French's Index of Differential Diagnosis*. 10th ed., 1973. Yearbook, Reprinted April, 1976. ($49.95)
Illich - *Medical Nemesis*. Pantheon, 1976. ($8.95)
Krupp - *Current Medical Diagnosis and Treatment*. 16th Annual Revision, Lange, 1977. ($16.00)
Thorn - *Harrison's Principles of Internal Medicine*. 8th ed. McGraw-Hill, 1977. ($37.00)

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MEETINGS

University of Colorado School of Medicine
Continuing Medical Education
INTENSIVE AND REHABILITATIVE RESPIRATORY CARE

University of Colorado Medical Center, Denver, Colorado

May 23-27, 1977

Contact: American College of Chest Physicians, 911 Busse Highway, Park Ridge, Illinois 60068.

* * *

FAMILY PRACTICE REVIEW
YMCA of the Rockies, Estes Park, Colorado

June 13-18, 1977
Credit: Approved for AMA Category 1-AAFP

* * *

ORTHOPEDICS AND PHYSICAL FITNESS FOR THE FAMILY PHYSICIAN
The Given Institute of Pathobiology, Aspen, Colorado

June 25-29, 1977
Credit: Approved for AMA Category 1-Application for AAFP prescribed credit.

* * *

63RD ANNUAL POSTGRADUATE COURSE IN OPHTHALMOLOGY AND SUMMER CONVENTION OF COLORADO OPHTHALMOLOGICAL SOCIETY
Snowmass-at-Aspen, Snowmass, Colorado

July 4-7, 1977
Credit: 12 hours AMA Category 1

* * *

PRACTICAL NEUROLOGY FOR THE INTERNIST AND FAMILY PHYSICIAN
The Given Institute of Pathobiology

July 11-13, 1977

* * *

GASTROENTEROLOGY: CURRENT CLINICAL TOPICS
The Gant, Aspen, Colorado

July 11-15, 1977
Credit: Approved for AMA Category 1 credit.

* * *

HEMATOLOGY-ONCOLOGY
The Given Institute of Pathobiology, Aspen, Colorado

July 14-17, 1977
Credit: Approved for AMA Category 1

* * *

GYNECOLOGY
The Mark, Vail, Colorado

July 17-20, 1977
Credit: Approved for AMA Category 1

INTERNAL MEDICINE
YMCA of the Rockies, Estes Park, Colorado

July 18-22, 1977
Credit: Approved for AMA Category 1

* * *

PEDIATRICS
The Given Institute of Pathobiology

July 24-27, 1977
Credit: Approved for AMA Category 1

* * *

PATHOLOGY IN PEDIATRICS
The Gant, Aspen, Colorado

July 25-29, 1977
Credit: Approved for AMA Category 1

* * *

PEDIATRIC DERMATOLOGY
The Given Institute of Pathobiology, Aspen, Colorado

July 27-29, 1977

Approved: For AMA Category 1

* * *

PATHOLOGY IN GYNECOLOGY AND OBSTETRICS
The Given Institute of Pathobiology, Aspen, Colorado

August 6-10, 1977
Credit: Approved for AMA Category 1

* * *

CLINICAL OTOLARYNGOLOGY FOR THE PRIMARY CARE PHYSICIAN
The Mark at Vail, Colorado

August 7-10, 1977
Credit: 26 hours Category II for Physician's Recognition Award-AMA

* * *

DISEASES OF THE KIDNEY AND RENAL FAILURE
The Given Institute of Pathobiology

August 10-13, 1977
Credit: Approved for AMA Category 1
Contact: Office of Postgraduate Medical Education, University of Colorado Medical Center, Container C295, 4200 East 9th Avenue, Denver, Colorado 80262

* * *

The Children's Hospital
PEDIATRIC EAR, NOSE AND THROAT SYMPOSIUM

Tammen Auditorium, The Children's Hospital, Denver, Colorado

May 27, 1977, 8 am-4 pm

Contact: Betsy Block, Department of Continuing Education, The Children's Hospital, 1056 East 19th Avenue, Denver, Colorado 80218, or Call: (303) 861-8888, Ex. 2280.

American Society for Clinical Oncology
SPECIAL MEETING

Denver Hilton, Denver, Colorado

May 16-17, 1977
Contact: Mrs. Jane Barnd, Charles B. Slack, Inc., Thorofare, New Jersey 08086.

The American Academy of Medical Directors
SECOND NATIONAL CONFERENCE OF MEDICAL DIRECTORS AND CHIEF MEDICAL EXECUTIVES

Camelback Inn, Scottsdale, Arizona

May 25-28, 1977
Contact: The American Academy of Medical Directors, 20 S. Quaker Lane, Alexandria, Virginia 22314

The National Intravenous Therapy Association, Inc.
5TH ANNUAL CONVENTION

Marriott Hotel, New Orleans, Louisiana

June 5-9, 1977
Contact: Susan Y. Thomason, 2202 W. Nash Rd., Apt. B-8, Wilson, N.C.

The American College of Cardiology
ECHOCARDIOGRAPHY: Fundamentals and New Developments in Cardiac Ultrasound

Town and Country Hotel, San Diego, California

June 7-10, 1977

TUTORIALS IN THE TETONS: CARDIAC EMERGENCIES
Jackson Lake Lodge, Grand Teton National Park, Moran, Wyoming

August 21-23, 1977
Contact: Miss Mary Anne McInerny, American College of Cardiology, 9650 Rockville Pike, Bethesda, Maryland 20014.

University of Utah Hospital Medical Center

COURSE FOR PRACTICING PHYSICIANS

University of Utah Hospital Medical Center, Salt Lake City, Utah.

June 20-22, 1977
Contact: James R. Swenson, MD, P.O. Box 2604, Salt Lake City, Utah 84110.

Wyoming Postgraduate Allergy Course, Inc.
21ST ANNUAL WYOMING POSTGRADUATE COURSE IN ALLERGY AND IMMUNOLOGY

Teton Village Resort, Wyoming

June 24-30, 1977
Credit: Approved for 36 hours credit by AAFP and AMA Continuing Education
Contact: Russell I. Williams, MD, 414 east 23rd Street, Cheyenne, Wyoming 82001.

Symposia de Santa Fe
SELECTED TOPICS IN HEMATOLOGY AND ONCOLOCY

La Fonda Hotel, Santa Fe, New Mexico

July 14-16, 1977

* * *

HYPERALIMENTATION AND FLUID THERAPY FOR SURGEONS

Inn at Loretto, Santa Fe, New Mexico

August 11-13, 1977

* * *

RADIOLOGY FOR FAMILY PRACTITIONERS

Hilton Inn, Santa Fe, New Mexico

September 14-16, 1977

Contact: Walter J. Levy, MD, P.O. Box 5175, Coronado Station, Santa Fe, New Mexico 87502.

The American Academy of Family Physicians, New Mexico Chapter
20TH ANNUAL RUIDOSO FAMILY PRACTICE SEMINAR

Inn of the Mountain Gods, Mescalero, New Mexico

July 18-21, 1977
Credit: 17 hours AAFP Prescribed, and 17 hours Category 4, New Mexico Board of Medical Examiners.
Contact: Bob Reid, P.O. Box 456, Sunland Park, New Mexico 88063.

Beth Israel Hospital
ASPEN MUSHROOM CONFERENCE

Hotel Jerome, Aspen, Colorado

August 7-12, 1977
Credit: AMA Physician Recognition Award, Up to 30 hours Category II.
Contact: Aspen Mushroom Conference, **c/o Beth Israel Hospital, 1601 Lowell Blvd., Denver, Colorado 80204.**

American Association of Diabetes Educators
4TH ANNUAL MEETING/EDUCATIONAL WORKSHOP

Hilton Hotel, Denver, Colorado

August 14-16, 1977
Contact: Kate Doyle, AADE, 3553 W. Peterson Ave., Chicago, Illinois 60659.

THE BOOK CORNER

New books received are acknowledged in this section and such acknowledgment must be regarded as sufficient return for the courtesy of the sender. Selection will be made for review in the interests of our readers and as space permits. Books are listed with advance data supplied by publishers. Prices quoted are not guaranteed. For further information, address queries to the publishers. Books here listed are available for lending from the Denver Medical Society Library.

The February 18, 1977 issue of **Legislative Roundup** *contains a concise summary of the A.M.A-sponsored "Comprehensive Health Care Insurance Act of 1977" (H.R. 1818-S.2'8.) Copies may be requested from the Library.*

Recent Acquisitions

Biliary Tract Surgery and Cholangiography: Vernon L. Stephenson. Springfield, Ill., Thomas, 1973. 186 p. $11.50

Breast Feeding and the Mother: Symposium on Breast Feeding and the Mother, London, 1976. N.Y., Elsevier, 1976. 280 p. (Ciba Foundation symposium, 45) Gift.

CT Scanners: American Hospital Association. Chicago, A.H.A., 1977. 134 p. $9.50.

Cardiac Pacemakers: Harold Siddons and Edgar Sowton. Springfield, Ill., Thomas, 1974. 331 p. $12.75.

Communicable and Infectious Diseases: Franklin H. Top. 8th ed. St. Louis, Mosby, 1976. 807 p. $39.50.

Consumer Directory of Medical Care for Northwestern Metropolitan Denver: Joel Karlin, ed. Westminster, Clear Creek Valley Medical Society, 1977. 264 p. $3.00. Gift.

Development of Professional Standards Review for Hospital Social Work: American Hospital Association. Chicago, A.H.A., 1977. 52 p. $7.00.

Doctors of the Old West: Robert F. Karalevitz. Seattle, Superior Publishing , 1967. 192 p. $8.95. Gift of Dr. Nolie Mumey.

The Frontal Lobes and Human Behavior.: John F. Fulton. Springfield, Ill., Thomas, 1972. 22 p. $4.50.

Geriatric Psychiatry: Leopold Bellak and Toksoz B. Karasu. N.Y., Grune & Stratton, 1976. 312 p. $17.50.

Health: A Victim or Cause of Inflation?: Michael Zabkoff, ed. N.Y., Prodist, 1976. 402 p. $9.60.

Hospital Management Systems: Montague Brown and Howard Lewis. Germantown, Md., Aspen Systems Corp., 1976. 305 p. $19.95.

Human Sterilization: Ralph M. Richart and Denis J. Prager, ed. Springfield, Ill., Thomas, 1972. 402 p. $21.00.

Hypernasality: Raymond Massengill. Springfield, Ill., Thomas, 1972. 140 p. $11.25.

Hyperthyroidism: Joel I. Hamburger. Springfield, Ill., Thomas, 1972. 198 p. $9.50.

The Influences of Pancreatic Tumors on the Stomach: Robert M. Zollinger and David W. Coleman. Springfield, Ill., Thomas, 1974. 205 p.

The Menstrual Cycle: Rudolf F Vollman. Philadelphia, Saunders, 1977. 193 p. (Major Problems in Clinical Obstetrics and Gynecology, v.7) c.$15.00.

The Nature of Occupational Cancer: Bertram D. Dinman. Springfield, Ill., Thomas, 1973. $8.75.

New Directions in Public Health Care; An Evaluation of Proposals for National Health Insurance: Institute for Contemporary Studies. San Francisco, Calif., I.C.S., 1976. 277 p. Gift.

A New Perspective on the Health of Canadians: Marc Lalonde. Ottawa, Ministry of National Health and Welfare, 1974. 152 p. Gift.

Physician's Current Procedural Terminology: Asher J. Finkel, ed. 4th ed. Chicago, A.M.A., 1977. 498 p. $12.00.

The Portal Venous System: Charles Marks. Springfield, Ill, Thomas, 1973. 137 p. $12.75.

Psychophysiological Aspects of Skin Disease: Francis A. Whitlock. Philadelphia, Saunders, 1976. 248 p. (Major Problems in Dermatology, v.8) $18.00.

The Pupil: Keith M. Zinn. Springfield, Ill, Thomas, 1972. 134 p. $8.50.

Rehabilitation of the Facially Disfigured: J.J. Longacre. Springfield, Ill, Thomas, 1973. 124 p. $8.50.

Respiratory Distress Syndrome of Shock and Trauma: Frank W. Blaisdell. Philadelphia, Saunders, 1977. 237 p. (Major Problems in Clinical Surgery, v.21) $15.00.

Risk Factors in Breast Cancer: Basil A. Stoll, ed. Chicago, Year Book, 1976. 269 p. $15.95.

Scar Tissue: J.J. Longacre. Springfield, Ill., Thomas, 1972 170 p. $14.75.

Small is Beautiful; Economics as if People Mattered: E.F. Schumacher. N.Y., Harper & Row, 1973. 290 p. $7.95.

Solid Liver Tumors: James H. Foster and Martin M. Berman. Philadelphia, Saunders, 1977. 342 p. (Major Problems in Clinical Surgery, v.22) c.$18.00.

Textbook of Gynecology: R.R. DeAlvarez. Philadelphia, Lea & Febiger, 1977. 546 p. $42.50.

Traumatic Injuries of the Heart and Great Vessels: Panagiotis N. Symbas. Springfield, Ill., Thomas, 1972. 188 p. $14.00.

Sex Change: Thomas Kando. Springfield, Ill., Thomas, 1973. 159 p. $7.50.

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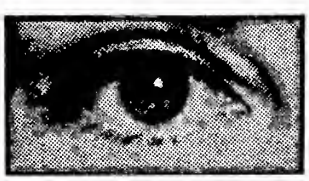

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July 14-16, 1977

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Snowmass, Colorado

MELANOMAS AND BIOMARKERS

Registration Fee: $25.00

Co-Sponsored by American Cancer Society, Colorado Division and
Colorado Medical Society

PROGRAM

THURSDAY, JULY 14, 1977

PRESIDING: *Richard M. Granberg, MD*

7:00 - 8:00 AM — Registration and Breakfast — Wildwood Inn

8:00 - 8:15 AM — Welcome
William R. Nelson, MD, President, Colorado Division, ACS
R. Lee Clark, MD, President, ACS
Carl H. McLauthlin, MD, President, Colorado Medical Society
John H. Budd, MD, President, American Medical Association

8:15 - 8:45 AM — Speaker: *Dr. Frank J. Rauscher*
Senior Vice President for Research
American Cancer Society
Topic: **The Cancer Problem**

8:45 - 9:05 AM — Speaker: *Henry M. Lewis,* MD
Dermatologist, Denver
Topic: **Diagnosis of Nevi and Melanomas**

9:05 - 10:15 AM — Speaker: *Richard J. Reed,* MD
Pathologist, Tulane University
Topic: **Cutaneous Malignant Melanoma: Its Evolution and Histologic Subtypes**

10:05 - 10:25 AM — Coffee Break — Opticon Lobby

10:25 - 11:25 AM — Speaker: *Harry Goldsmith,* MD
Surgeon, Temple University
Topic: **Current Concepts in the Treatment of Melanoma**

11:25 - 12:00 AM — Speaker: *R. Lee Clark,* MD
President, American Cancer Society

5:30 - 7:30 PM — Reception for faculty and attendees, guests
Cash Bar — Wildwood Inn

AMA Physician Recognition Award, Category 1 and AAFP Credit Applied For

For Further Program Information Contact:
American Cancer Society, Colorado Division, Inc.
1809 East 18th Avenue, Denver, Colorado 80218
Phone: (303) 321-2464

For Reservation Information
Contact:
Snowmass, Box 5566, Snowmass, Colorado 81615

Program planned so attendees can take advantage of various recreation Activities available at Snowmass and Aspen

FRIDAY, JULY 15, 1977

PRESIDING: *Joseph L. Kovarik,* MD

7:00 - 8:00 AM — Registration and Breakfast — Wildwood Inn

Speaker: *John H. Budd,* MD
President, American Medical Association

8:00 - 9:00 AM — Speaker: *Richard J. Reed,* MD
Topic: **Cutaneous Malignant Melanoma: Perameters Related to Prognosis**

9:00 - 9:50 AM — Speaker: *William R. Nelson,* MD
Topic: **Perfusion and the Surgical Treatment of Metastatic Melanoma**

9:50 - 10:05 AM — Coffee Break — Opticon Lobby

10:05 - 10:40 AM — Speaker: *Jack Speer,* MD
Medical Director, Penrose Cancer Hospital, Colorado Springs
Topic: **Systemic Treatment of Metastatic Melanoma**

10:40 - 11:15 AM — Speaker: *Paul Anderson,* MD
Penrose Cancer Hospital, Colorado Springs
Topic: **Adjuvant Immunotherapy in the Treatment of Melanoma**

11:15 - 12:00 AM — Panel: All guest faculty: *Paul Anderson,* MD, *Harry Goldsmith,* MD, *Henry Lewis,* MD, *Frank Rauscher,* MD, Richard Reed, MD, and *Jack Speer,* MD

Moderator: *Erick Ratzer,* MD

7:30 PM — Barbecue — Guests Welcome (Cost $7.00 per person including children)

SATURDAY, JULY 16, 1977

PRESIDING: *William R. Nelson,* MD

7:00 - 8:00 AM — Registration and Breakfast — Wildwood Inn

Speaker: *Frank J. Rauscher,* PhD
Topic: **What's Down the Road in Cancer Research**

8:00 - 8:30 AM — Speaker: *R. Lee Clark,* MD

8:30 - 9:15 AM — Speaker: *Stanley G. Korenman,* Md
Topic: **Endocrine Markers**

9:15 - 9:40 AM — Speaker: *Gene H. Moore,* Md
Penrose Cancer Hospital, Colorado Springs
Topic: **C.E.A. Biomarkers**

9:40 - 10:05 AM — Speaker: *Sylvia Kerr,* PhD
University of Colorado Medical Center, Denver
Topic: **Biochemical Markers for Cancer Diagnosis and Monitoring of Therapy**

10:05 - 10:20 AM — Coffee Break — Opticon Lobby

10:20 - 10:55 AM — Speaker: *David Cox,* MD
Urologist, Denver
Topic: **Recent Advances in Tumor Markers for Testicular Cancer**

10:55 - 11:20 AM — Speaker: *Stanley G. Korenman,* MD
Topic: **Estrogen Receptors**

11:20 - 12:00 AM — Panel: All guest faculty: *David Cox,* MD, *Sylvia Kerr,* PhD, *Stanley Korenman,* MD, *Gene Moore,* MD, and *Frank Rauscher,* PhD
Moderator: *Alexis Lubchenco,* MD

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FAMILY PRACTICE, Idaho Springs, Colorado. 30 miles West of Denver on I-70. Retiring doctor of 40 years practice desires quick sale of practice, equipment, large residence; 3 exam rooms, x-ray room, and reception room. Owner will finance. Contact: Eureka Realty Company, (303) 572-1565, or write: P.O. Box 280, Idaho Springs, Colorado, 80452. 377-7-1B

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OFFICE SPACE AVAILABLE: completely furnished office in three-man building. East Denver, near hospitals. Available on monthly basis or long-term lease. Contact: Howard Bronson, MD, 1425 Jackson Street, Denver, Colorado, 80206, or call: (303) 333-1577. 377-16-2B

WANTED: Position as Family Nurse Practitioner in Colorado mountains. RN, 14 years. Nurse Practitioner 1 year. NP Education: 9 months, Montana State Program of Rural Family Nurse Practitioners. Work hard for good salary. Request resume to P.O. Box 123, Albion, Nebraska 68620, or call: (402) 395-2551, after 6 pm. 377-18-2B

WANTED: Four General Practitioners or four Family Practitioners, one internist, one General Surgeon, one Pathologist, and one Radiologist needed in progressive rural area - in Clinic setting; new hospital completed in one year; excellent schools; recreation; servicing 15,000 to 25,000 people. Income open. Contact: Administrator, Frances Mahon Deaconess Hospital, Glasgow, Montana 59230 377-19-2B

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NEED HELP PLANNING OR LANDSCAPING the land surrounding your business or home? Possible drought conditions will be given special consideration. Call: 355-8486, Gail Barry & Associates, Women Landscape Designers and Contractors, Serving the East Denver Area. 377-25-3B

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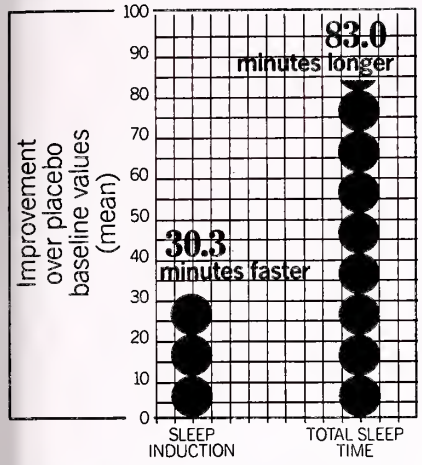
Elderly insomniacs fell asleep faster, slept longer[1]



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Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.,* operating machinery, driving).

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Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.,* excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

REFERENCES:
1. Frost JD Jr: Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
2. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
3. Robinson DS, Amidon EL: Interaction of benzodiazepines with warfarin in man, in *The Benzodiazepines,* edited by Garattini S, Mussini E, Randall LO. New York, Raven Press, 1973, p. 641

Objective evidence proves insomnia relief in elderly patients

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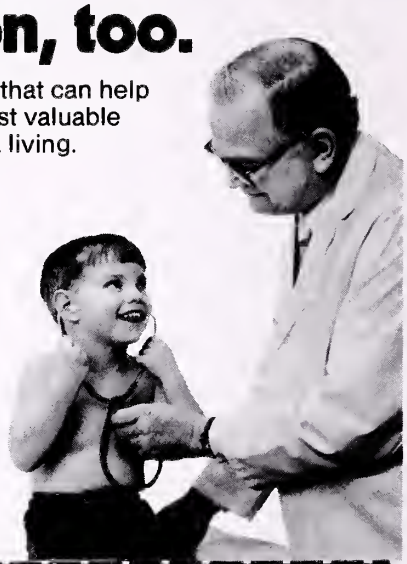

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Rocky Mountain Medical Journal

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July-August 1977
VOLUME 74 • NUMBER 4

SHELL FALLS on the western slope of the Big Horn Mountains, U.S. 14 between Greybull and Sheridan. These beautiful falls drop 75 feet into magnificent Shell Canyon.

If you've been prescribing pentobarbital or secobarbital for insomnia, there's good reason to reconsider.

More effective than secobarbital through 14 nights of administration...[1,2]

In two separate sleep laboratory studies,[1] secobarbital 100 mg was found to lose much of its initial hypnotic effect in insomniac subjects within a two-week administration period. Dalmane® (flurazepam HCl), however, has been proved[2] to remain effective for both inducing and maintaining sleep at the end of two weeks, with the usual adult dosage (30 mg *h.s.*). Elderly and debilitated patients should receive 15 mg initially, to help preclude oversedation, dizziness or ataxia.

And more effective than pentobarbital through 28 nights of administration...[3,4]

In an original study designed to evaluate hypnotic effectiveness for 28 consecutive nights of use, the relative ineffectiveness of pentobarbital was established after only two weeks.[3] Dalmane, however, remained effective not only for 14 nights, but for 28 nights in chronic insomniacs,[3,4] without increasing dosage from night to night. Prolonged administration of Dalmane is seldom necessary, but when it is, periodic blood counts and liver and kidney function tests should be performed.

More proven safety benefits for your patients than barbiturates...

Specific safety benefits not shared by barbiturate hypnotics: Dalmane (flurazepam HCl) may be used in patients on chronic warfarin therapy; no unacceptable fluctuation in prothrombin time has been reported.[5,6] And Dalmane has been proved not to interfere chemically with many common laboratory tests.[7-9] (Alterations have been reported due to pharmacological effects; see Adverse Reactions section of complete product information.)

Dalmane® (flurazepam HCl) C

30-mg and 15-mg capsules

Unsurpassed record of efficacy and safety

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:

Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.

Contraindications: Known hypersensitivity to flurazepam HCl.

Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.,* operating machinery, driving).

Usage in Pregnancy: **Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.,* excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.

Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.

Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

References:

1. Kales A, *et al*: *Clin Pharmacol Ther 20*: 541-545, Nov 1976
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9. Kaplan SA, *et al*: *J Pharm Sci 62*:1932-1935, 1973

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Klebsiella
Aerobacter
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Corynebacterium
Streptococcus
Pneumococcus

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Aerobacter
Escherichia

In vitro overlapping antibacterial action of Neosporin® Ointment (polymyxin B-bacitracin-neomycin).

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(Polymyxin B-Bacitracin-Neomycin)

Each gram contains: Aerosporin® brand Polymyxin B Sulfate 5,000 units; zinc bacitracin 400 units; neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); special white petrolatum qs; in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

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Rocky Mountain Medical Journal

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If the AMA didn't speak for the profession, who would?

Who would speak for the profession on the 2,500 health bills introduced in every Congress? Or the regulations issued by federal agencies?

Who would state the profession's views on national health insurance? Utilization Review Regulations? The Health Planning Act of 1974? Maximum Allowable Cost Regulations? Health Manpower?

Who would provide the scientific input and the practitioner's experience and knowledge so essential to legislation on drugs, cancer, heart disease, communicable diseases? Can you think of anyone?

The fact is, there is only one organization that can — and does — speak for the profession as a whole. The AMA.

It does so to protect the basic freedoms of medical practice in any federal health program that might be enacted; and even more important, to promote legislation for better health care for the entire public.

The AMA's voice can only be as strong as the members of the profession choose to make it. With your support, the AMA can be even more effective spokesman.

Join us.
We can do much more together.

Dept. of Membership Development
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535 N. Dearborn St./Chicago, IL 60610

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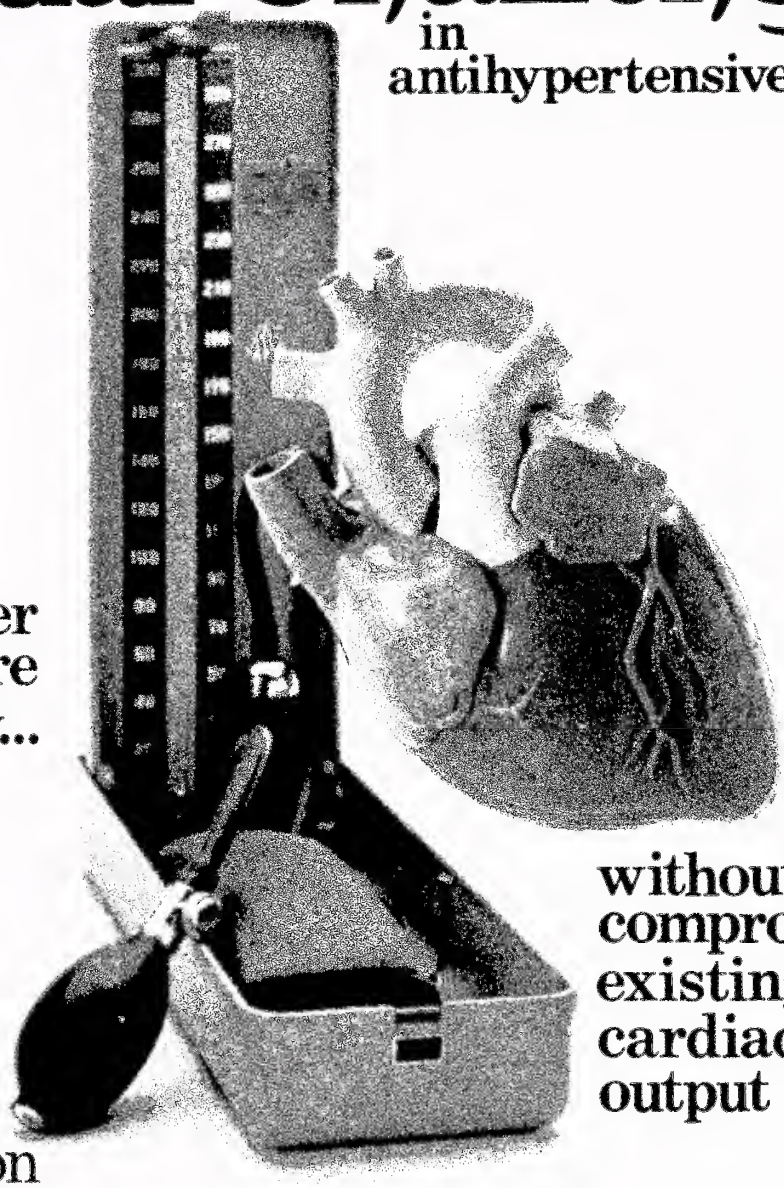
A Dual Challenge in antihypertensive therapy



to lower blood pressure effectively...

without compromising existing cardiac output

in hypertension

TABLETS: 250 mg, 500 mg, and 125 mg

ALDOMET® (METHYLDOPA | MSD)

helps lower blood pressure effectively... usually with no direct effect on cardiac function–cardiac output is usually maintained

ALDOMET is contraindicated in active hepatic disease, hypersensitivity to the drug, and if previous methyldopa therapy has been associated with liver disorders.
It is important to recognize that a positive Coombs test, hemolytic anemia, and liver disorders may occur with methyldopa therapy. The rare occurrences of hemolytic anemia or liver disorders could lead to potentially fatal complications unless properly recognized and managed. For more details see the brief summary of prescribing information.

For a brief summary of prescribing information, please see following page.

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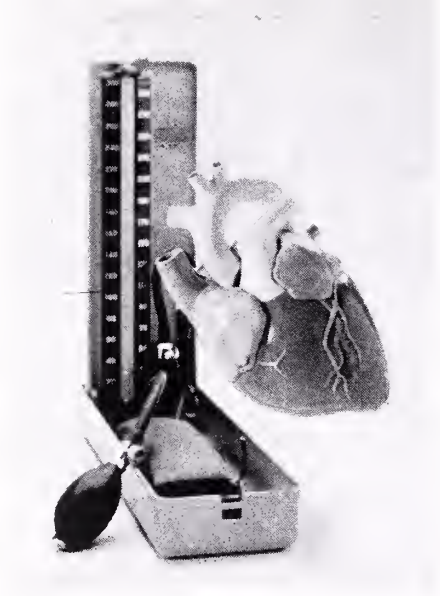

in hypertension

ALDOMET®

(METHYLDOPA|MSD)

helps lower blood pressure effectively... usually with no direct effect on cardiac function– cardiac output is usually maintained

Contraindications: Active hepatic disease, such as acute hepatitis and active cirrhosis; if previous methyldopa therapy has been associated with liver disorders (see Warnings); hypersensitivity.

Warnings: It is important to recognize that a positive Coombs test, hemolytic anemia, and liver disorders may occur with methyldopa therapy. The rare occurrences of hemolytic anemia or liver disorders could lead to potentially fatal complications unless properly recognized and managed. Read this section carefully to understand these reactions.

With prolonged methyldopa therapy, 10% to 20% of patients develop a positive direct Coombs test, usually between 6 and 12 months of therapy. Lowest incidence is at daily dosage of 1 g or less. This on rare occasions may be associated with hemolytic anemia, which could lead to potentially fatal complications. One cannot predict which patients with a positive direct Coombs test may develop hemolytic anemia. Prior existence or development of a positive direct Coombs test is not in itself a contraindication to use of methyldopa. If a positive Coombs test develops during methyldopa therapy, determine whether hemolytic anemia exists and whether the positive Coombs test may be a problem. For example, in addition to a positive direct Coombs test there is less often a positive indirect Coombs test which may interfere with cross matching of blood.

At the start of methyldopa therapy, it is desirable to do a blood count (hematocrit, hemoglobin, or red cell count) for a baseline or to establish whether there is anemia. Periodic blood counts should be done during therapy to detect hemolytic anemia. It may be useful to do a direct Coombs test before therapy and at 6 and 12 months after the start of therapy. If Coombs-positive hemolytic anemia occurs, the cause may be methyldopa and the drug should be discontinued. Usually the anemia remits promptly. If not, corticosteroids may be given and other causes of anemia should be considered. If the hemolytic anemia is related to methyldopa, the drug should not be reinstituted. When methyldopa causes Coombs positivity alone or with hemolytic anemia, the red cell is usually coated with gamma globulin of the IgG (gamma G) class only. The positive Coombs test may not revert to normal until weeks to months after methyldopa is stopped.

Should the need for transfusion arise in a patient receiving methyldopa, both a direct and an indirect Coombs test should be performed on his blood. In the absence of hemolytic anemia, usually only the direct Coombs test will be positive. A positive direct Coombs test alone will not interfere with typing or cross matching. If the indirect Coombs test is also positive, problems may arise in the major cross match and the assistance of a hematologist or transfusion expert will be needed.

Fever has occurred within first 3 weeks of therapy, sometimes with eosinophilia or abnormalities in liver function tests, such as serum alkaline phosphatase, serum transaminases (SGOT, SGPT), bilirubin, cephalin cholesterol flocculation, prothrombin time, and bromsulphalein retention. Jaundice, with or without fever, may occur, with onset usually in the first 2 to 3 months of therapy. In some patients the findings are consistent with those of cholestasis. Rarely fatal hepatic necrosis has been reported. These hepatic changes may represent hypersensitivity reactions; periodic determination of hepatic function should be done particularly during the first 6 to 12 weeks of therapy or whenever an unexplained fever occurs. If fever and abnormalities in liver function tests or jaundice appear, stop therapy with methyldopa. If caused by methyldopa, the temperature and abnormalities in liver function characteristically have reverted to normal when the drug was discontinued. Methyldopa should not be reinstituted in such patients.

Rarely, a reversible reduction of the white blood cell count with primary effect on granulocytes has been seen. Reversible thrombocytopenia has occurred rarely. When used with other antihypertensive drugs, potentiation of antihypertensive effect may occur. Patients should be followed carefully to detect side reactions or unusual manifestations of drug idiosyncrasy.

Use in Pregnancy: Use of any drug in women who are or may become pregnant requires that anticipated benefits be weighed against possible risks; possibility of fetal injury can not be excluded.

Precautions: Should be used with caution in patients with history of previous liver disease or dysfunction (see Warnings). May interfere with measurement of: uric acid by the phosphotungstate method, creatinine by the alkaline picrate method, and SGOT by colorimetric methods. Since methyldopa causes fluorescence in urine samples at the same wavelengths as catecholamines, falsely high levels of urinary catecholamines may be reported. This will interfere with the diagnosis of pheochromocytoma. It is important to recognize this phenomenon before a patient with a possible pheochromocytoma is subjected to surgery. Methyldopa is not recommended for patients with pheochromocytoma. Urine exposed to air after voiding may darken because of breakdown of methyldopa or its metabolites.

Stop drug if involuntary choreoathetotic movements occur in patients with severe bilateral cerebrovascular disease. Patients may require reduced doses of anesthetics; hypotension occurring during anesthesia usually can be controlled with vasopressors. Hypertension has recurred after dialysis in patients on methyldopa because the drug is removed by this procedure.

Adverse Reactions: *Central nervous system:* Sedation, headache, asthenia or weakness, usually early and transient; dizziness, lightheadedness, symptoms of cerebrovascular insufficiency, paresthesias, parkinsonism, Bell's palsy, decreased mental acuity, involuntary choreoathetotic movements; psychic disturbances, including nightmares and reversible mild psychoses or depression.

Cardiovascular: Bradycardia, aggravation of angina pectoris. Orthostatic hypotension (decrease daily dosage). Edema (and weight gain) usually relieved by use of a diuretic. (Discontinue methyldopa if edema progresses or signs of heart failure appear.)

Gastrointestinal: Nausea, vomiting, distention, constipation, flatus, diarrhea, mild dryness of mouth, sore or "black" tongue, pancreatitis, sialadenitis.

Hepatic: Abnormal liver function tests, jaundice, liver disorders.

Hematologic: Positive Coombs test, hemolytic anemia. Leukopenia, granulocytopenia, thrombocytopenia.

Allergic: Drug-related fever, myocarditis.

Other: Nasal stuffiness, rise in BUN, breast enlargement, gynecomastia, lactation, impotence, decreased libido, dermatologic reactions including eczema and lichenoid eruptions, mild arthralgia, myalgia.

Note: Initial adult dosage should be limited to 500 mg daily when given with antihypertensives other than thiazides. Tolerance may occur, usually between second and third month of therapy; increased dosage or adding a thiazide frequently restores effective control. Patients with impaired renal function may respond to smaller doses. Syncope in older patients may be related to increased sensitivity and advanced arteriosclerotic vascular disease; this may be avoided by lower doses.

How Supplied: Tablets, containing 125 mg methyldopa each, in bottles of 100; Tablets, containing 250 mg methyldopa each, in single-unit packages of 100 and bottles of 100 and 1000; Tablets, containing 500 mg methyldopa each, in single-unit packages of 100 and bottles of 100.

For more detailed information, consult your MSD representative or see full prescribing information. Merck Sharp & Dohme, Division of Merck & Co., Inc., West Point, Pa. 19486 J6AM07(707)

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methylprednisolone, Upjohn

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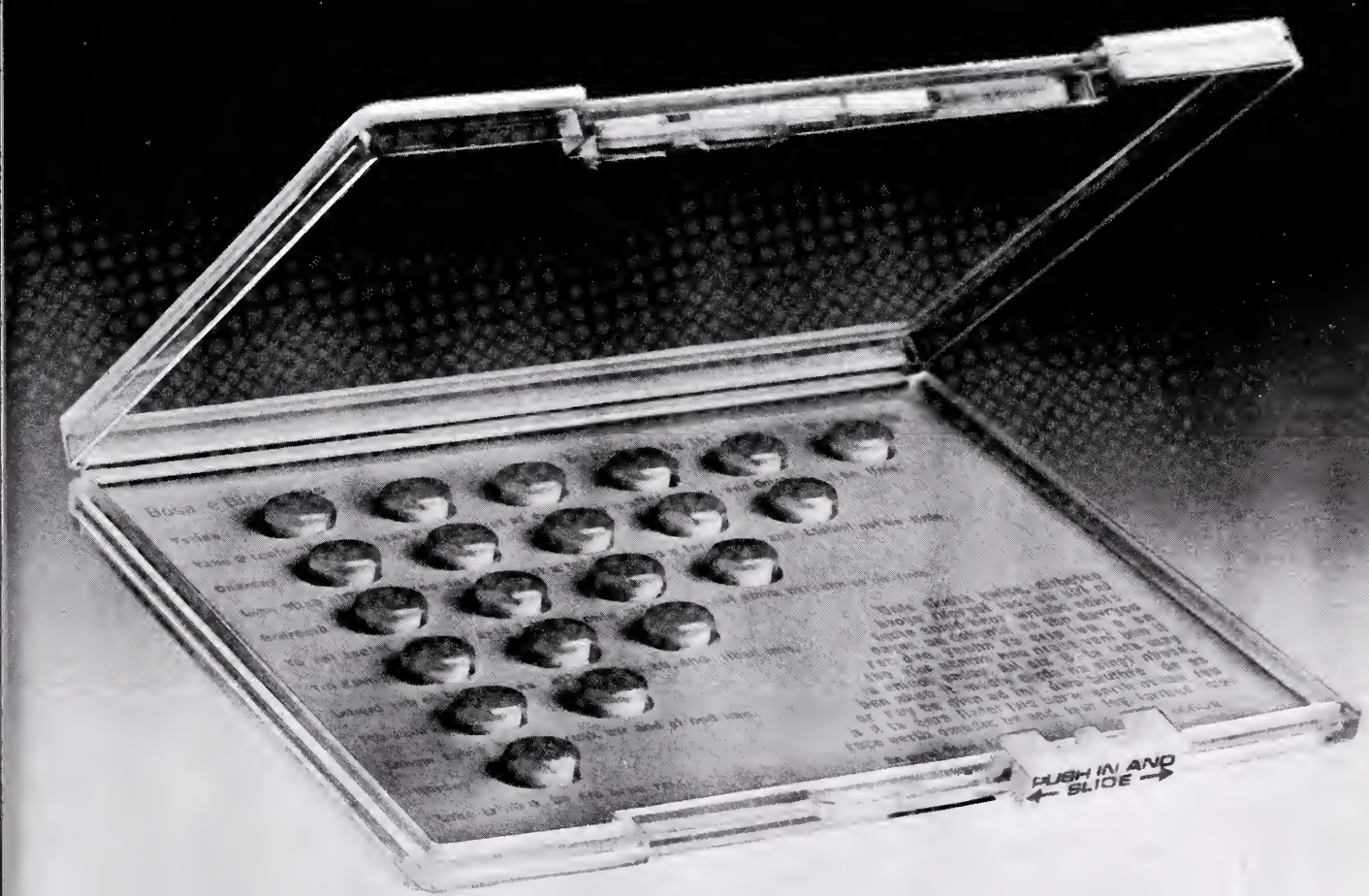

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MILITANTS VEXED AT PRIVACY.

Wanted Movies of Ceremony, But Both Factions Are

WASHINGTON, Aug. 26, 1920—

Social Security Bill Is Signed, Gives Pensions to Aged, Jobl

Roosevelt Approves Message Intended to Benefit 30,0 Persons When States Adopt Cooperating Laws—He a the Measure 'Cornerstone' of His Economic Progra

SENATE APPROVES 18-YEAR OLD VOTE IN ALL ELECTIONS

Amendment to Constitution is Sent to House, Where Passage is Expected

WASHINGTON, March 10, 1971—The Senate approved today, 94 to 0, and sent to

WASHINGTON, Aug. 1, The Social Security Bill, po a broad program of unemplo insurance and old age in and counted upon to bene 20,000,000 persons, becam day when it was signed b dent Roosevelt in the pre those chiefly responsible o ting it through Congress.

Mr. Roosevelt called the "the cornerstone in a s which is being built that means comple

TRUMAN CLOSES TED NATIONS CONFEREN ITH PLEA TO TRANSLATE CHARTER INTO DEEDS

NEW WORLD HOPE

President Hails 'Great Instrument of Peace,' Insists It Be Used

HISTORIC LANDMARK

Meeting Gives Standing Ovation as Executive Pictures Peace Gain

"If we fail to use it," he declared to the solemn final meeting of the delegates, 'we shall betray all of those who have died in order that we might meet here in freedom and safety to create it.'

"If we seek to use it selfishly—for the advantage of any one nation or any small group of nations—we shall be equally guilty of that betrayal."

Fervent Interpolation

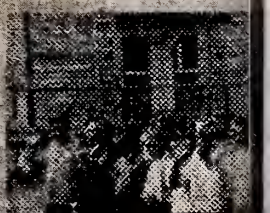
The President, speaking in the auditorium of the War Memorial Opera House, built in memory of sons of the Golden Gate city who gave their lives in the first World War, in which he himself served, seemed to give unconscious expression to the solemn feeling of the occasion when, at the outset of his speech, he interpolated the words, half a hope, half a prayer:

"Oh, what a great day this can be in history!"

Just before the plenary session the President accompanied the eight United States delegates to

SIGNI the Draf Ends No

WASHINGTON, Jan. 27, 1973—"With the signing of the peace agreement in Paris today, and after receiving a report from the Secretary of the Army that he foresees no need for



PATIENT PACKAGE INSERTS: A CONCEPT WHOSE TIME HAS COME?

The consumer's right to know is an irreversible and desirable trend of the Seventies. It extends, and properly, to a patient's right to know more about his or her prescription medications. One way, gaining favor, is through patient package inserts. Wisely-prepared and properly distributed when medically indicated, they could markedly improve patient knowledge and drug therapy—laudable goals by anyone's standards.

The PMA endorses these goals and will work with government, the health professions and consumers to achieve them.

The Advantages

The concept holds promise of benefits: better patient understanding of the product prescribed, better adherence to the treatment plan, and more awareness of possible side reactions.

Every doctor has had patients who fail to finish antibiotic regimens because they feel better. Some patients assume that if one tranquilizer or analgesic is good, two may be twice as good. Still others fail to report dizziness while on antihypertensive therapy —and so on.

Problems like these might arise less often if the patient received written information in addition to verbal instructions. Some studies suggest that patients are more receptive to such materials, and they more often understand the verbal instructions and follow them, when inserts are used.

The Disadvantages

There are also some potential problems. Obviously, the inserts must be clearly phrased, without extraneous or complex detail. How much information is enough? How can it be kept current? Should all patients receive the same information? Should inserts be included with all drugs? Should only potential problems be listed or are patients better off with a "fair balance" presentation that describes usefulness as well as drawbacks?

These and similar questions require answers, since model inserts have yet to be properly developed and tested. Despite the need for these studies, the FDA is proceeding prematurely with inserts on selected products. We think the Congress is the only place where the matter can be given the proper legal status and direction, particularly since it represents a conceptual change in the legal, medical and social framework of the nation's prescription drug information system.

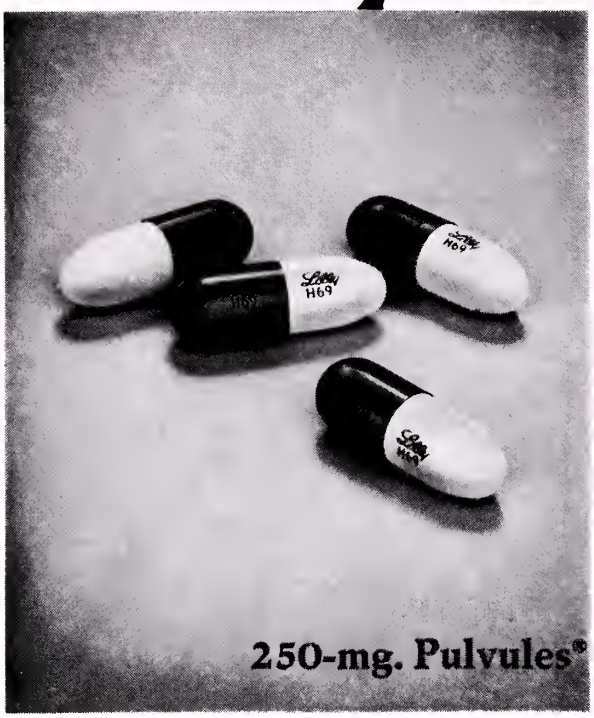
The Solution

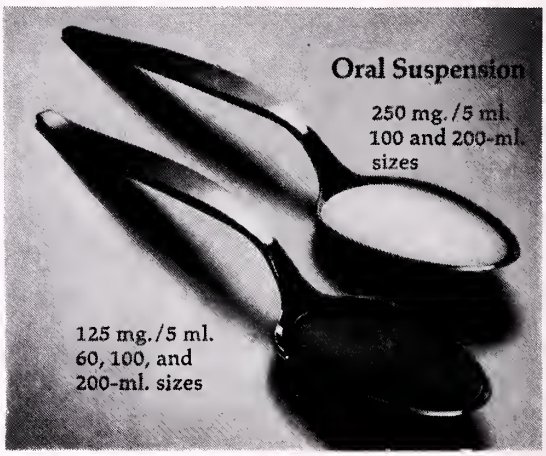
The PMA believes that carefully-devised pilot studies of various kinds of inserts are needed. They should be developed and implemented with full participation by doctors, pharmacists, consumers, communications experts and the drug industry. Such studies will provide reliable pathways to follow, so that inserts will be useful aids to medical practice.

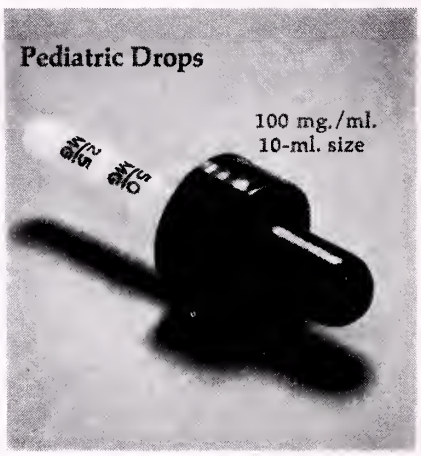
And particularly we think that you should be closely involved in this debate and in these studies and decisions. Otherwise, people with less experience and qualifications may control the purposes, content and use of a tool with considerable promise for improved patient care. It could make a difference in your practice tomorrow, and more importantly, in the health of your patients.

PMA
THE PHARMACEUTICAL MANUFACTURERS ASSOCIATION
1155 FIFTEENTH ST., N. W., WASHINGTON, D. C. 20005

easy to take







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A Time for Reassessment

Do you feel like you are being pulled, pushed, poked and shoved from all directions? Well, relax, you are. From all directions come the attacks—unnecessary surgery, exorbitant fees, fraud, increasing cost of medical care, failure to provide care to all segments of our society, and on and on. I, as you, have grumbled and fussed about these multiple assaults.

Then I started thinking. If so many people claim there to be so many problems directly and indirectly involving us as physicians, maybe there is *some* truth in what they are saying.

So, I am urging you and me to reconsider our common position, which is to practice the *art* and *science* of medicine, to perform our duties at the least cost possible to our patients, to listen to our adversaries and attempt to correct our professional deficiencies, and, finally, to actively support and improve our local, state, and national professional societies. For it is only with our house in order, with a united front and with innovative leadership that we, as a profession, can survive and continue to do what we are trained for - to take care of people.

James E. Stoetzel, MD.
President
Wyoming State Medical Society

"If The Oath Fits, Wear It!"

Like presidential addresses of clubs and societies, medical editorials seem to hew to such a rigid format that one hesitates to begin. The form and substance of editorials to be read by one's medical colleagues may be pre-cast in concrete more than any others. As readers of medical journals know, the editorial must view with varying degrees of alarm activities of anyone outside the medical profession who threatens our territorial boundaries. The federal government has always brought forth the loudest alarm, third party carriers the next greatest collection of decibels, and then, of course, the lesser threats include everything from malpractice insurors to one-eyed hospital administrators.

The other cardinal principle in the editorial content of a medical journal is always to point with pride to the attributes and only hint obliquely at some of the faults of the medical brethren, and then only if absolutely necessary. Never speak or write as openly as you would after two beers. For example, the editorialist should never comment on his colleagues as "he's a guy that ought to be watched", "he hasn't read a medical journal since the Civil War", or "that bum shouldn't operate on a dog", etc... Anyone can see, these type quotations should never be used, because a healthy majority of the readers would immediately conclude that they were intended to be the victim of the verbal onslaught. There are many other no-no's that inhibit the freedom of editorialists but, on the other hand, allow for his longevity. Having been around awhile, it is not my intention consciously to make any of the above mistakes.

One of the few comforting things about our troubles of today is to perceive, on looking back in history, that practically every generation has had almost identical problems. This repetition runs the gamut from the fear of socialized medicine to the problems in raising our kids, alcoholism and divorce in our friends, watching a business-oriented physician fleece his patients, and so on. It is comforting to realize that these problems have been with us for a long time, as have been the many warnings issued from all sides. On the other hand, everyone has to make his own judgments as to where we are and where we are going. After all, the prophets of doom may have been right all along and this last warning may be followed by the predicted holocaust. However, we should be well advised to recall an oath we took at one time:

I swear by Apollo the physician, and Asclepius, and Health, and All-heal, and all the gods and goddesses, that, according to my ability and judgment, I will keep this Oath and this stipulation — to reckon him who taught me this Art equally dear to me as my parents, to share my substances with him, and relieve his necessities if required; to look

upon his offspring in the same footing as my own brothers, and to teach them this art, if they shall wish to learn it, without fee or stipulation; and that by precept, lecture, and every other mode of instruction, I will impart a knowledge of the Art to my own sons, and those of my teachers, and to disciples bound by a stipulation and oath according to the law of medicine, but to none others.

I will follow that system of regimen which, according to my ability and judgment, I consider for the benefit of my patients, and abstain from whatever is deleterious and mischievous. I will give no deadly medicine to any one if asked, nor suggest any such counsel; and in like manner I will not give to a woman a pessary to produce abortion. With purity and with holiness I will pass my life and practice my Art.

"I will not cut persons labouring under the stone, but will leave this to be done by men who are practitioners of this work. Into whatever houses I enter, I will go into them for the benefit of the sick, and will abstain from every voluntary act of mischief and corruption; and, further, from the seduction of females or males, of freemen and slaves.

"Whatever, in connexion with my professional practice, or not in connexion with it, I see or hear, in the life of men, which ought not to be spoken of abroad, I will not divulge, as reckoning that all such should be kept secret. While I continue to keep this oath unviolated, may it be granted to me to enjoy life and the practice of the art, respected by all men, in all times! But should I trespass and violate this Oath, may the reverse be my lot!"
Oath of Hippocrates

If the oath fits, wear it!

Francis A. Barrett, MD
Wyoming Editor
Rocky Mountain Medical Journal

Stand Up and Fight

Once in every young boy's life he is faced with the neighborhood bully, or another young boy whom he doesn't know in an alley or on a playground and is challenged to his first "stand up and fight." Chances are, the young man is not happy about the situation in which he finds himself, but sooner or later he realizes that if he backs down the remainder of the school year, or the summer, or the camp, will be miserable. Looking into the next few months, it is clear that the physicians of our country are going to be much in the predicament of our young man ... that is, challenged to defend their way of practice and way of life.

The President, the Administration and its myriad of federal agencies, and the Congress are beating their drums and organizing their courage for an all-out revamping of the health care delivery system as we know it. The banner under which this foray will be made is "cutting waste and holding down the spiraling costs of health care." The result could be a staggering bureaucracy which is administering an impossible program, which deprives your patients of improved care, robs you of the freedom to provide the best care possible to the patients who have chosen you, and places innumerable irritations and obstacles in the path of all health care workers who are interested in performing their tasks in a professional manner.

Again, like the young man with his physical challenge, doctors have three choices. First, there is total capitulation — an unpalatable opportunity at best. Second, there is blind opposition — probably more emotionally rewarding, but not very productive in the final analysis. Finally, there is the opportunity, within some rapidly changing limits, to choose the nature and grounds on which the confrontation will take place.

The initial step probably should be to examine the ring of onlookers, and it is apparent that not too many of the observers are truly "on our side." Therefore physicians must, first and foremost, count on themselves for most of the help. It would also be prudent for each physician to examine closely what his priorities are and what changes he would welcome or consider inconsequential and what principles and practices are unalterable. The next logical step involves communications — to translate the individual physician's thoughts into group priorities which can be represented by county, state and national medical organizations.

Moving from internal communications, the profession's first public "round" must be to communicate its story to the people. It must be readily apparent to all the consuming groups in our nation that the health care industry is accomplishing much, is responding to the citizen's pleas for better care, and is willing to accept intelligent changes which will eliminate needless duplication and create a climate where progress can be made. In the 1977 AMA Leadership Conference, three British physicians recommended two basic tenets to our medical leadership: (1) Don't allow physicians to become employees of government, and (2) Don't allow the establishment of a giant bureaucracy to administer a national health insurance program. Perhaps, these are starting points for physicians in establishing their "ring strategy."

Finally, each physician, individually and through his organizations, must take part in an effort to mold upcoming legislation on cost control and national health insurance. The truly frightening part of this coming "fight" is that through apathy and a "don't get involved in medical politics" attitude, a segment of the medical profession will "throw in the towel" for all doctors before physicians have their chance to defend themselves in the ring of public opinion and national legislation.

Robert Smith
Executive Director
Wyoming State Medical Society

The Restive Syndrome

You have a middle-aged man in room 314 recovering slowly from his first acute coronary, and he has complicating dysrhythmias. You are worried about a young widow in room 206 showing signs of losing her ability to walk from multiple sclerosis. Have the two patients anything in common? Indeed they have! Both tend to give you the Restive Syndrome...an uneasy feeling in back of your mind that something is wrong over and apart from the medical complications—but it isn't clear what.

Both patients are breadwinners for their families. Both stand a good chance of being unable to return to what earned them and their families a decent living. It's the latter element that gives rise to the Restive Syndrome—the sensation that perhaps the tough problems for both of these patients may just be starting.

Sure, you think of rehabilitation in both cases, and this usually means a referral to the rehabilitation department in the hospital, where certainly both patients are given the best chance to learn to accommodate to their respective ailments. But that's only a beginning.. The next step you take may be crucial. What about their support background? Do they have workman's compensation? Will it pay off in this case? If so, for how long? How long will it take them to get Federal social security aid? Will it sustain the family? These are but a few pertinent questions it won't take long to answer. And if the answers aren't particularly enlightening, you may want to take it a step farther and ask what one thing might help both these people the most, beyond your good medical offices, of course. It may be that calling your local Vocational Rehabilitation agency will help relieve the burden of this uneasiness you feel.

Vocational Rehabilitation, VR for short, is the oldest helping agency in the Nation, predating the entire gamut of Social Security and Industrial Compensation agencies. It remains today as state government's single *anti-welfare* agency, since its basic goal is restoring major handicaps and retraining those with them to be productive useful people. In fact, productive work is a *must* goal for all VR clients. The most recent figures from around the country indicate VR programs return $9.00 for every $1.00 spent on behalf of clients. This adds up to a whopping multi-million dollar return to your community in terms of tax dollars saved in welfare costs and in the return flow of income to local economies.

Every state in the union has a state VR agency, efficiently managed by a cadre of counselors who are at once psychologists, occupational specialists, job facilitators, family planners, paramedics and all-around helpful people. I have been around state government for some time and have yet to see a more dedicated group of folks do so much with the limited budgets, the almost impossible demands and the Herculean tasks which are set before them. That is not to say that VR is always successful in what it tries to do, or that VR never makes mistakes. We in VR are both unsuccessful and mistaken at times primarily because of the complexities of the human organisms with which we work. The point is, we keep trying.

And VR wouldn't get to first base were it not for another group of loyal and extremely talented people - a small but energetic group of physicians in every state who act as VR consultants. They maintain medical vigilance on all VR patients as they progress to the ranks of the able and employed.

So the next time you are afflicted with the Restive Syndrome concerning a seriously ill or incapacitated patient, think of the VR agency. It may cure your problem and help with those of your patients.

J. Frank Hurdle, MD.
Chief Medical Consultant
Division of Rehabilitation
State of Colorado

Eosinophilic fasciitis*

Robert R. Kanard, MD, Cheyenne, Wyoming

At the Pan American Congress on rheumatic diseases in June, 1974, Dr. Lawrence Shulman presented the first two cases of a disease which a year later would be called eosinophilic fasciitis and which now has the eponym of Shulman's Syndrome. These patients, both men, had a history of unusual physical exertion followed by illness characterized by high sedimentation rate, peripheral eosinophilia ranging from twelve to thirty-seven per cent, and an elevated IgG. Rheumatoid factor, L. E. cell preparations, and anti-nuclear antibodies were normal. Biopsy of the fascia showed an intense infiltration with plasma cells and lymphocytes both about and separate from blood vessels, at times in follicles, but eosinophils were absent. In 1975, Rodnan presented seven cases at the annual meeting of the American Rheumatism Association, and Caperton and Hathaway presented five more cases. The criteria were refined and changed somewhat. I shall present a young female whose illness has been diagnosed Eosinophilic Fasciitis but who is particularly interesting in that she also has organs involved that are associated with progressive systemic sclerosis.

CASE REPORT

A 39-year-old white female presented in the office January 14, 1975, with a history of minor trouble with arthritis in small joints for several years. She could always control the discomfort with aspirin, the arthralgia being intermittent and of little significance. However, in November, 1974, she increased the amount of aspirin to 10 or 12 tablets a day, not because of an increase in joint pain, but because of a severe increase in fatigue and tiredness. The increased amount of Excedrin and aspirin lessened joint pain, but not the fatigue. By January 6, 1975, her ankles began to swell, and this had become her greatest concern. At this initial visit, sedimentation rate was 109, latex agglutination was negative, hemoglobin was 11.2 grams and hematocrit was 33. She was admitted to the hospital on January 16, 1975, for diagnosis and treatment.

On physical examination, her weight was 135 pounds, blood pressure was 110/60, pulse was 108. She was normally developed, well nourished and in no distress. She gave a history suggesting Raynaud's Phenomenon, in that her hands became bluish or blanched when exposed to cold, causing her to wear gloves almost constantly when outside in the winter. Her hands flushed red when warmed. Her skin was tender, edematous, indurated and tight in the forearms, lower legs, and less so in the hands and feet. The joints were slightly tender to palpation; there was no synovitis of any joints. Eyes, ears, nose, throat, lungs, heart, and abdomen were not remarkable. The lower extremities showed four plus firm ankle and leg edema from the knees down. The dorsalis pedis, popliteal and femoral pulses were present and bilaterally equal and strong. Posterior tibial pulses were somewhat decreased.

Laboratory work included an admission CBC, which showed 10.7 grams of hemoglobin, hematocrit of 34, 4,900 WBC with sixty-six per cent segmented neutrophils, eighteen per cent lymphocytes, four per cent eosinophils, nine per cent bands, one monocyte and one basophil. On one differential count in the hospital, eosinophils numbered six per cent of the total. Reticulocyte count was one per cent, cell indices were normal except the mean corpuscular volume was slightly elevated to 103. Immunoglobulin study showed IgG 3200 (normal is 710 to 1540), IgA 564 (normal is 60 to 490), IgM 412 (normal is 42 to 261). Serum complement was 2100 (normal is 125 to 165). The following tests or studies were normal or negative: urinalysis, sodium, potassium, chloride, CO_2 calcium, phosphorous, BUN, creatinine, SGOT, LDH, CPK, alkaline phosphatase, L. E. cell preparation twice, latex agglutination and chest film. Creatinine clearance was normal at 126 c.c. per minute. A.N.A. titer was positive, 1:80. A barium swallow with thin barium was normal but when it was repeated with thick barium, a definite lack of peristaltic activity was demonstrated, compatible with the findings seen in early sclerodermatous involvement of the esophagus. Roentgengram of the right

*Presented at WSMS Winter Seminar, January, 1977.

hand showed slight tissue loss over the tips of the index and fifth fingers, again, a finding consistent with scleroderma, but not diagnostic. EKG showed right bundle branch block, and pulmonary function studies were normal. Skin biopsy showed only atrophic changes, which were compatible with, but not diagnostic of scleroderma.

Her hospital course was unremarkable, except for afternoon temperature of 101.6 the first two days; the third day her temperature fell to 100.6, and then she became afebrile. She was relatively comfortable in the hospital. She was believed to have early scleroderma in the edematous stage. She was started on Bufferin, 8 to 12 tablets per day, and was discharged on January 25, 1975, to be followed in the office.

From the time of discharge to her readmission in June, 1975, she really did not improve. She was treated with aspirin and later Motrin. However, she continued to have four plus leg edema, extremely edematous forearms, and fatigue. Tiredness remained severe to the point that she was thinking about giving up her job as a teacher. On May 8, 1975, she weighed 127 pounds, 12 pounds less than on her first visit in January, 1975, and at the time of her readmission she weighed 120 pounds. Weight loss was thought to represent true protein loss, since the edema remained about the same.

She was readmitted on June 16, 1975, specifically for a biopsy of the skin, subcutis, fascia and muscle, to rule out eosinophilic fasciitis. Her physical findings had not changed significantly. She was still anemic with 8.9 grams of hemoglobin. White count was 6900 with a normal differential, with only two per cent eosinophils, and an absolute eosinophil count of 99 cells per cubic centimeter. Sedimentation rate was still elevated to 96. Because of proximal muscle weakness, an SGOT, LDH and CPK were obtained and were normal. Her serum iron was low at 42. Latex agglutination was negative; the A.N.A. titer was negative; skin biopsy showed minimal changes; muscle was normal; the fascia showed an intense and heavy infiltration of leukocytes and fibroblastic proliferation. The leukocytes included neutrophils, mononuclear cells with a moderately prominent number of eosinophils. It was thought she had eosinophilic fasciitis, and at this time she was started on 40 mgs of Prednisone, to be followed in the office.

She was seen ten days after discharge from the hospital and stated she was feeling much better generally, noted a significant increase in her pep and energy and noted less ankle edema. Her arm swelling had also decreased, and the leg edema had dramatically improved. On July 17, 1975, her hemoglobin had risen to 12 grams, her hematocrit was 36, her sedimentation rate had dropped to 42. By August 28, 1975, her arms and legs had essentially returned to normal, and her pep and energy was normal, she was doing her housework, looking forward to teaching, and had actually been hiking with her family in the mountains through the summer. At that time her hemoglobin was 13.8 grams, her hematocrit was 43, her sedimentation rate had dropped to 21. She has continued to improve up to present, with a gradual reduction of her Prednisone to 10 mgs every other day. Her last hemoglobin was 15.5 grams and hematocrit was 45 in December, 1976. At that time her sedimentation rate was 35.

Discussion

Shulman's original description was presented as cases of diffuse fasciitis with hypergammaglobulinemia and eosinophilia, and was thought to represent a new syndrome.[1] He presented two male patients, 53 and 19 years old, with a diffuse scleroderma-like illness with firm, taut skin bound down to underlying structures and sparing the face in both of his patients and the hands and feet in one. Flexion contractures of the elbows and knees with limitation of abduction at the shoulders developed a few weeks after onset. Raynaud's Phenomenon, myositis and visceral manifestations of systemic sclerosis were absent. Both patients had elevated sedimentation rates and hypergammaglobulinemia, with elevation of the IgG portion. Rheumatoid factor, L. E. cell preparations, antinuclear antibodies, complement, cryoglobulins were all negative or normal. Biopsies showed striking thickening of the fascia between the subcutis and muscle. Within the thickened connective tissue, there was an infiltration with plasma cells and lymphocytes; however, eosinophils were absent. Skin biopsies showed no changes of scleroderma, and muscle biopsies were normal. The first patient responded dramatically and completely to Prednisone therapy; however, the second patient reversed his laboratory abnormalities, but clinical improvement was minimal. Both these patients had a history of unusual physical exertion before the onset of illness, a historical fact which has not persisted in the history of the twelve patients presented a year later.

A year later these criteria were reiterated. Rodnan's seven cases (five male and two female) were 11 to 60 years old, first noted pain, swelling and tenderness of the distal portions of the forearm and/or legs, and soon thereafter developed severe induration of the skin and subcutaneous tissues with marked limitation of motion of hands and feet. In two cases the lower abdomen was affected, and in another, the face. None had Raynaud's Phenomenon or evidence of visceral manifestations characteristic of systemic sclerosis. The blood eosinophil count ranged from 416 to 2,725 cells per cubic centimeter, and eight to twenty-nine per cent eosinophils on the differential. IgG was increased in six; A.N.A. including anti-RNA were negative; complement was normal. Biopsies obtained from forearm or leg showed sclerosis of the dermis and inflammation and fibrosis of the panniculus and deep fascia, the latter being greatly thickened and heavily infiltrated by lymphocytes, plasma cells, histiocytes, and eosinophils. Improvement occurred in six treated with Prednisone and the seventh had a spontaneous remission. In Caperton's five

cases, he suggested that the disease was an unusual steroid-responsive variant of scleroderma or diffuse morphea. The five patients, two female and three male, range in age from 4 to 46 years old. All five patients had sudden onset of illness with skin edema, followed by striking "hide-bound" appearance, relative sparing of the skin of face and fingers, patches of localized morphea on the trunk, striking eosinophilia, hypergammaglobulinemia and the IgG portion elevated, rapid progressing with contracture of wrists, elbows, shoulders; synovial inflammation was observed in three. On biopsy, the prominent changes are fibrosis with inflammation, often perivascular, in the deeper layers of the subcutaneous tissue and extending into the muscle fascia. Early inflammatory cells including eosinophils were present in the dermis. Both Rodnan's and Caperton's patients had eosinophils in the fascia, while Shulman's original cases explicitly mention the absence of eosinophils. Rheumatoid factor was present in two patients and an A.N.A. titer was positive in one patient.

The patient presented fulfills several of the criteria of eosinophilic fasciitis: (1) sudden onset, (2) rapid progression, (3) marked edema of the forearms and lower legs, and (4) lesser involvement of the hands and feet. She also had: (5) a mild eosinophilia on differential, and (6) elevated sedimentation rate, (7) elevated immunoglobulin G, and (8) eosinophils in her biopsy. Scleroderma has as some of its characteristics Raynaud's Phenomenon, elevated sedimentation rate, anemia, positive or negative A.N.A. titer and latex agglutination, hypergammaglobulinemia, edema and round cell infiltrate with fibroblastic proliferation of the dermis and subcutis, and a decrease in esophageal contractions and motility. These also are problems which our patient manifested in addition to the characteristic findings of eosinophilic fasciitis. Although this patient fulfills the criteria for eosinophilic fasciitis, she has several of the components of scleroderma. Besides those already mentioned, she had biopsy changes and x-ray changes of the hands, compatible with early scleroderma.

It is important to consider eosinophilic fasciitis in patients who present with scleroderma or appear to have early progressive systemic sclerosis, because this is the only condition to date that can be successfully treated with medication. This is in sharp contrast to systemic sclerosis. The use of corticosteroids in systemic sclerosis is actually felt to be contraindicated, because it is thought to potentiate the onset of renal disease and malignant hypertension, resulting in the rapid deterioration and death of the patient. Eosinophilic fasciitis, in contrast, responds dramatically to corticosteroids, and recently has been treated with other anti-inflammatory agents with subsequent improvement.

In conclusion, a patient with eosinophilic fasciitis has been presented, with signs associated with progressive systemic sclerosis. This patient responded dramatically to corticosteroid therapy, and at this time remains well and fully active without pain or active disease. It is felt that the clinician should consider this disease whenever the diagnosis of scleroderma is being entertained, because eosinophilic fasciitis can very closely mimic the early stages of scleroderma. However, in constrast to scleroderma, eosinophilic fasciitis is treatable and responds dramatically in most cases.•

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Elevated serum calcium

R. Dale Liechty, MD*, Denver

Dramatic new developments in calcium and vitamin D research promise exciting new treatments for patients with related disorders. The first part of this paper concerns hypercalcemia and the second discusses current concepts of calcium metabolism.

Hypercalcemia

CAUSES: Disseminated cancers and hyperparathyroidism are the leading causes of hypercalcemia. Sarcoidosis, milk alkali syndrome, hyperthyroidism, vitamin D poisoning, ectopic production of PTH from some tumors, especially lung cancer, and thiazide ingestion also cause hypercalcemia.

Some key words - *bones, stones,* abdominal *groans,* psychic *moans* and fatigue *overtones* - help us remember the clinical picture of hypercalcemia (if we also think of polyuria and polydipsia). The abdominal groans come from duodenal ulcers and pancreatitis. The psychic symptoms can, of course, be subtle and difficult to differentiate in our world of neuroses.

MARGINAL ELEVATION: What should be done if a calcium is only slightly elevated at 11 mg%? The Mayo Clinic conducted a five-year study of outpatients with minimally elevated calciums and no symptoms. Within this period twenty per cent of patients required operation because they became symptomatic, another twenty per cent were lost to followup, and sixty per cent remained asymptomatic. With an asymptomatic elderly patient, for example, with a serum calcium of 10.5 or 11 mg%, one is justified in simply watching the patient, as long as there are no symptoms or renal problems. If the calcium rises suddenly, renal problems can result, sometimes rapidly. Serum calciums remain the single most important diagnostic test for hyperparathyroidism. There has been nothing in the past decade to alter reliance on this test. If there are two or three elevated serum calciums (above 10.5 mg% in our laboratory) and other causes of hypercalcemia are ruled out, the diagnosis is almost always hyperparathyroidism. Parathormone assays are less reliable, but they have been extremely helpful in some cases. With these two tests diagnosis can be made in about ninety-five per cent of all patients. The serum levels of phosphorus and the tubular reabsorption of phosphate are less reliable indicators of hyperparathyroidism.

TREATMENT: Serum levels of calcium above 14 mg% indicate a medical emergency. Most to be feared is nephrocalcinosis or coma. The three leading therapies are intravenous phosphorus - given orally, if the patient is awake - salmon calcitonin and, most important of all, diuresis. Parathyroid adenomas sometimes demand emergency operation. If the patient has diffuse cancer, mithramycin is the most valuable drug (25μg qid, a total of 100 mg/day). This drug probably acts on the osteoclasts in the bone where it suppresses calcium release. Finally, for vitamin D intoxication or sarcoidosis, cortisone can be extremely helpful. Cortisone has been used for a number of years for hypercalcemia, but the mechanism remains obscure.

A decade or two ago it was taught that eighty-five per cent of all hyperparathyroidism was caused by an adenoma. Despite much recent controversy, most authorities still accept this figure. The other fifteen per cent have as a cause hyperplasia, multiple adenomata or a combination of the two. From a surgical standpoint, if there is a single adenoma, no fam-

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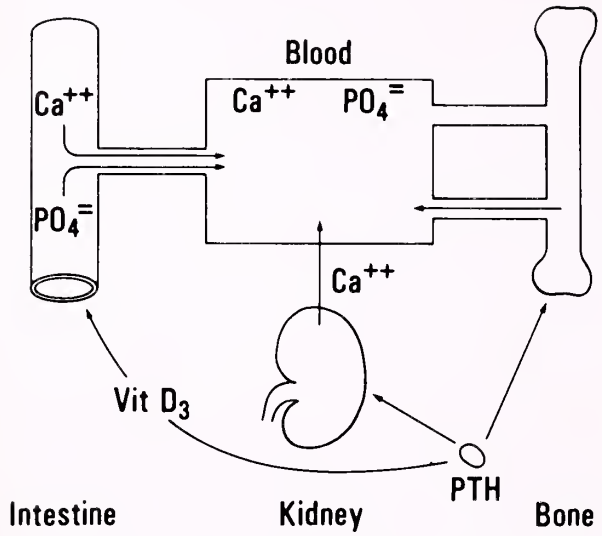

Fig. 1. Parathormone - Ca^{++} and PO_4. PTH has a direct effect on bone and renal **tubule** *cells to increase the blood concentration of Ca^{++}. PTH indirectly affects the gut by stimulating the renal parenchymal cells to secrete 1,25(OH)2 vitamin D_3, the potent polar metabolite of vitamin D_3. This agent increases the absorption of both Ca^{++} and PO_4 from the gut.*

ily history of hypercalcemia, no multiple endocrine adenopathies and normal renal function, we simply excise the adenoma after biopsy of one other normal gland. However, if there is a family history of hypercalcemia or related endocrine syndromes, if there is a renal cause of the disease or if more than one gland is enlarged, we remove three and one-half glands.

Calcium Metabolism

Most of us were taught that $Ca^{++} \times PO_4$ equals a constant, but no one a decade or two ago knew exactly why. This product is still useful, in that when it approaches the 60-70 range, dangerous calcification is seen. Below 20, we see failure of calcification. Let us consider the main agents that control calcium and phosphorus metabolism.

PARATHORMONE: We have long been taught, and many of the current textbooks continue to teach, that parathormone (PTH) directly affects three areas: GI tract, kidney, and bone. We can still keep this concept, but now we understand the mechanism more clearly. Notice that parathormone goes to the kidney and the kidney changes vitamin D_3 to its

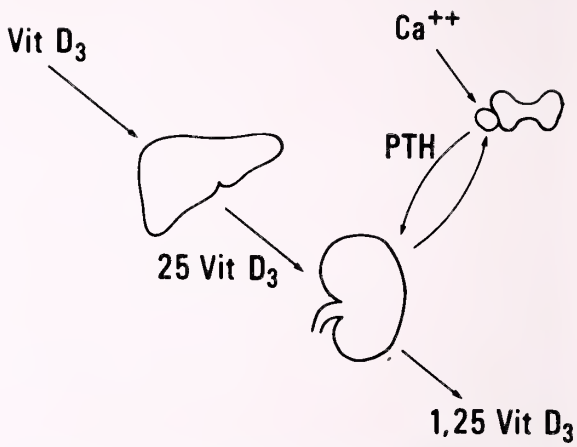

Fig. 2. Vitamin D_3 - a hormone. The renal parenchymal cells convert vitamin D_3 to 1,25(OH)2 vitamin D_3 under the influence of two stimuli: (1) elevated PTH, or (2) lowered serum PO_4. PTH and 1,25(OH)2 vitamin D_3 form a feedback, control system. The kidney is the endocrine organ and 1,25(OH)2 vitamin D_3 the hormone.

metabolically active end products (Fig. 1). Remember that parathormone switches on the active metabolite of vitamin D_3 and this in turn affects the gut. Parathormone in itself has no direct effect on intestinal absorption of calcium. Overall, parathormone has a direct effect on bone and kidney, and an indirect effect on the gut, a secondary effect, through stimulation of the final active metabolite of vitamin D_3. In the bone, parathormone by itself can cause calcium release. It increases the reabsorption of calcium from the kidneys and has a phosphaturic effect.

VITAMIN D - A HORMONE: Vitamin D is now accepted by scientists all over the world to be a hormone, a new and most important concept (Fig. 2). A hormone is a substance produced in one gland that has its effect elsewhere in the body. Almost all hormone systems have feedback relationships that regulate their production. Vitamin D_3 taken orally is absorbed into the serum where it then enters the liver. There it is hydroxylated to the 25-vitamin D_3 form - 25-hydroxy-vitamin D_3. It then proceeds to the kidney, where another hydroxyl group is added to form 1,25-dihydroxy-vitamin D_3. This metabolically active end product has a feedback relationship with parathormone. PTH stimulates the renal parenchymal cells to convert 25(OH) vitamin D_3 to 1,25(OH)2 vitamin D_3. In turn, 1,25(OH)2 vitamin D_3, either directly or

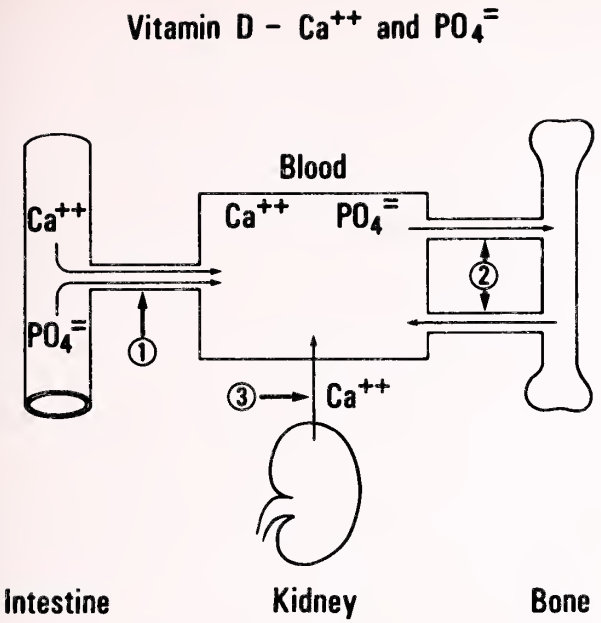

Fig. 3. Vitamin D_3 - Ca^{++} and PO_4. 1,25(OH)$_2$ vitamin D_3 acts on: (1) the gut to increase absorption of both Ca^{++} and PO_4; (2) bone where normally it provides a saturated solution of Ca^{++} and PO_4 to nourish bone - but, working with PTH, it can also release Ca^{++} from bone; and (3) kidney (probably) to increase Ca^{++} reabsorption.

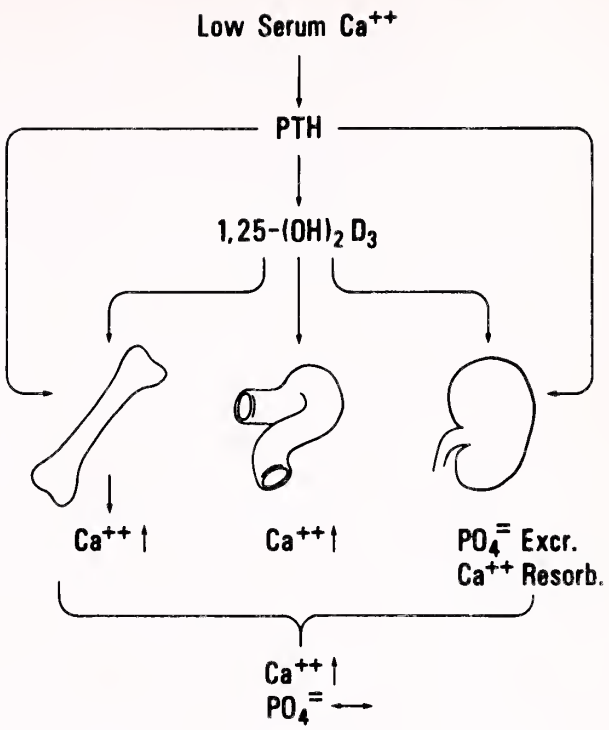

Fig. 4. Low Serum Ca^{++} control system. Working in unison PTH and 1,25(OH)$_2$ vitamin D_3 return low Ca^{++} to normal without raising PO_4 (see text).

indirectly, by way of calcium and phosphorus, turns off PTH. This is the feedback relationship and the endocrine organ is the kidney. These active metabolites may prove to be the drugs of choice for renal bone disease, osteoporosis and other diseases of calcium metabolism. Research in this area may represent the most dramatic single advance in medicine in the past decade, according to some researchers.

Now let us see how vitamin D_3 works (Fig. 3). Vitamin D_3, as mentioned, stimulates the absorption of phosphorus and calcium from the gut. Parathormone incites the renal cells to convert the 25(OH)$_2$ vitamin D_3 to the more active 1,25(OH)$_2$ form. Low PO_4 levels also stimulate this conversion. Second, vitamin D has an effect on bone. Paradoxically, it can move calcium and phosphorus into or out of bone. Vitamin D_3, according to Deluca, provides a super-saturated solution to nourish bone and keep it calcified at all times. This is its primary function. But it has to act in the presence of parathormone to remove calcium. The function in the kidneys is less certain but probably it helps reabsorb calcium to some extent. So these two hormones, parathormone and vitamin D_3, work in concert to keep blood levels of phosphorus and calcium normal and to increase them in times of emergency. Calcium, especially, vitally affects enzymatic processes in cells.

Ca^{++} AND PO_4 CONTROL SYSTEMS: Since vitamin D_3 increases absorption of both calcium and phosphorus from the gut, how can a low serum calcium correct itself without raising serum phosphorus? Figure 4 gives us the explanation: Low serum calcium turns on PTH. PTH, as mentioned, goes to the kidney and switches on the final metabolic form of vitamin D_3, which is so potent, the 1,25 form. Also, PTH has a direct effect on bone and kidney. Note that 1,25(OH)$_2$ vitamin D_3 has an effect on all three organs. Parathormone itself has no direct effect on the gut; it is mediated via vitamin D_3. So calcium and phosphorus rise in the blood because of the dual action of vitamin D_3 and PTH. Calcium and phosphorus are absorbed in the gut because of the action of vitamin D_3. In the kidney the excess amount of PTH causes phosphaturia. Calcium is reabsorbed in proportionally greater amounts, and so the final effect is that calcium rises but PO_4 remains the same.

Low serum PO_4 corrects itself by a different mechanism (Fig. 5). Two stimuli turn on the conversion of 1,25(OH)$_2$ vitamin D_3 in the kidney. One is parathormone. The other is low serum phosphorus. But because there is no excess PTH at the time, calcium or phosphorus

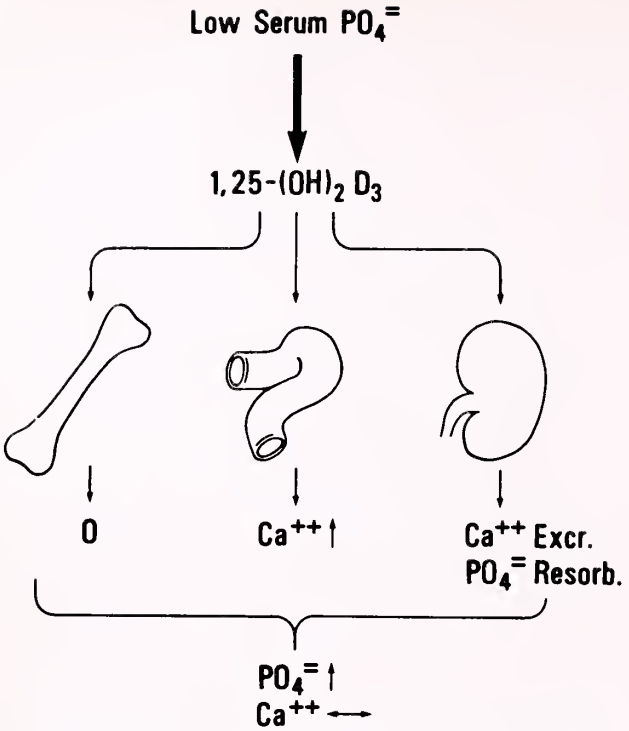

Fig. 5. Low serum PO_4 control system. When serum PO_4 drops, 1,25(OH)₂ vitamin D₃ returns it to normal without elevated Ca^{++} (see text).

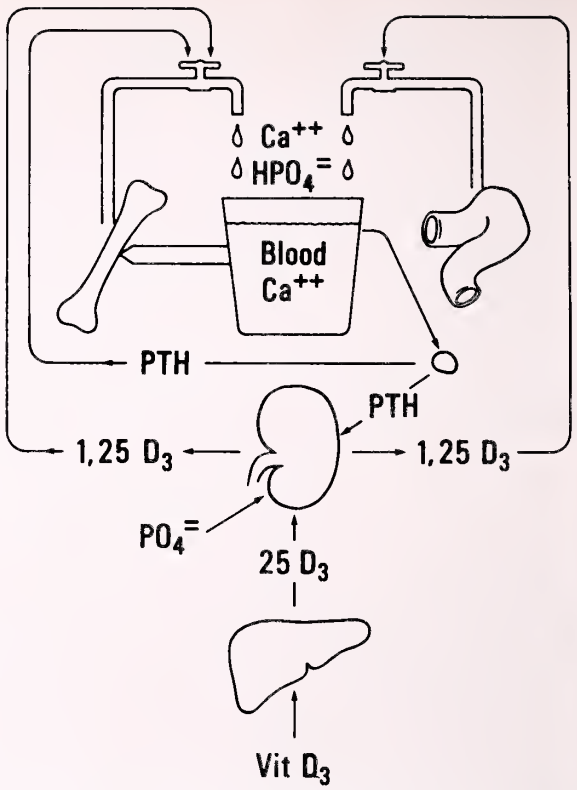

Fig. 6. Control systems for Ca^{++} and PO_4. Calcitonin (not shown) acts alone on bone, to prevent release of Ca^{++} (see text).

cannot leave the bone. The 1,25(OH)₂ vitamin D₃ form goes to the gut and strongly increases absorption of calcium and phosphorus. Because there is no excess PTH in this mechanism, calcium is excreted in larger amounts and phosphorus is reabsorbed. (PTH, of course, causes excretion of phosphorus.) So phosphorus rises in the blood, and calcium remains the same. These are the self-correcting mechanisms for keeping blood phosphorus and calcium normal. The first one is a dual mechanism involving both PTH and 1,25(OH)₂ vitamin D₃. The latter is a single mechanism, featuring only 1,25(OH)₂ vitamin D₃.

DISEASES THAT AFFECT CA^{++} AND PO_4 METABOLISM: In terms of these new concepts in calcium and phosphorus metabolism, some common diseases illustrate all of these mechanisms. Vitamin D is taken in by mouth. The liver changes it to 25-vitamin D₃. The kidney, under two stimuli - either parathormone, coming from the parathyroid gland, or decreased levels of phosphorus - turns on the conversion of vitamin D to its active 1,25(OH)₂ vitamin D₃ form. PTH and 1,25(OH)₂ vitamin D₃ move to the bone and increase calcium and phosphate absorption from the bone. Vitamin D, having been stimulated either by parathormone or by low serum phosphorus levels, moves to the gut and increases the absorption of both calcium and phosphorus. *Rickets* or *osteomalacia* (adult rickets) features a simple lack of vitamin D. This was, of course, discovered in the late 1920s when Steinbach found that irradiating foods could produce vitamin D. *Diseases of the liver* can cause altered vitamin D metabolism and bone disease. Perhaps thirty per cent of patients on long-term *Dilantin-phenobarbital* treatment will have significant bone disease, chiefly osteoporosis. Anticonvulsant therapy apparently interferes with the hydroxylation mechanism in the liver. *Biliary cirrhosis* also inhibits the 25-hydroxylation of vitamin D₃ in the liver. Both of these diseases respond to 25(OH)² vitamin D₃ which is available now experimentally (Calcifediol).

In *hyperparathyroidism,* the parathyroid glands secrete too much PTH, autonomously. PTH in turn stimulates the secretion of 1,25(OH)₂ vitamin D₃. It opens up the faucets from bone and gut. Again, the 1,25 form of vitamin D₃ is put out in high amounts because both PTH and low phosphorus stimulate its production by renal cells. As mentioned at the beginning, we treat this with two agents: phosphorus and calcitonin. Increased levels of phosphorus

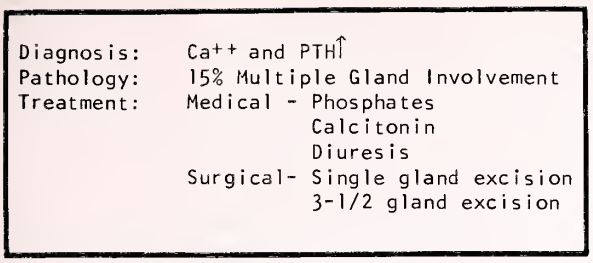

Fig. 7. Hyperparathyroidism. Summary of the laboratory diagnosis, pathology and treatment.

turn off the renal mechanism. Calcitonin acts on bone to prevent calcium and phosphorus release (Fig. 6).

In the opposite disease, *hypoparathyroidism,* the PTH falls and PO_4 rises. Since both stimuli are absent, there is no stimulation of 1,25(OH)$_2$ vitamin D_3 production by the renal cells and no stimulation of gut absorption of $Ca^{++}$and PO_4.

Treatment with vitamin D increases the intestinal absorption of calcium and phosphates, and this is the only route to maintaining normal serum Ca^{++}levels. Ideally, 1,25(OH)$_2$ vitamin D_3 is the best treatment because it acts rapidly and can be withdrawn rapidly if the calcium rises above normal. Vitamin D preparations available now must be given in huge doses to produce the same effect (up to 200,000 units; 400 units/day is normal). So 1,25(OH)$_2$ vitamin D_3 provide a more precise and safe method of treating hypoparathyroidism.

In terms of numbers of patients, *osteoporosis* is certainly the most important disease of calcium metabolism. Some scientists think osteoporosis is a defect in the 1-hydroxylation mechanism in the kidney. Animal studies substantiate these theories. Even more important, three clinical studies of osteoporosis treated with 1,25(OH)$_2$ vitamin D_3 show improvement in bone densitometry, bone mass, bone histology and in patient's symptoms. If future studies confirm these early ones, we may have the first really effective treatment for this common

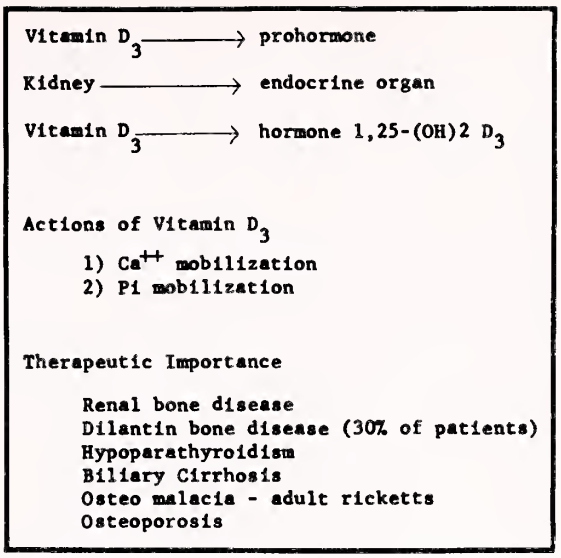

Fig. 8. Vitamin D_3 metabolism. Summary showing that vitamin D_3 fits the definition of a hormone with an essential role in health and important therapeutic possibilities in disease.

malady. Certainly there will be more about these exciting new discoveries in vitamin D metabolism in future years.

Summary

Fig. 7 summarizes the important aspects of hyperparathyroidism. Serum calcium and parathormone levels allow us to confirm the diagnosis in about ninety-five per cent of cases.

Despite conflicting reports in the literature, about eighty-five per cent of all cases feature a single adenoma that responds to excision. In the remainder, multiple gland involvement demands excision of three and one-half glands.

Fig. 8 summarizes new information concerning vitamin D and calcium metabolism. Acting as an endocrine organ, the kidney converts vitamin D substrates to its active metabolic end product - 1,25(OH)$_2$ vitamin D_3. This substance has exciting possibilities in the treatment of some common diseases that have frustrated physicians for years.●

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Emergency medical services

Current and Future Status in Wyoming

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"An Emergency Medical Service's delivery system is as strong as it's weakest link. As data on acute myocardial infarction and multiple trauma have been accumulated over the past several years, it has become clear that a considerable portion of morbidity and mortality occurs outside the hospital before the patient reaches medical care. It has similarly become clear that implementation of high standards of advanced pre-hospital life support at the scene, and in transport, can improve the outcome of critically ill and injured patients, thus reducing the need for prolonged, often futile, terminal intensive care."[1]

In Wyoming, the implementation of high standards of pre-hospital life support began several years ago. In 1971, a National Registry of Emergency Medical Technicians, or EMTs, was created, with the support of the AMA, utilizing private funds—an attempt to standardize, on a national level, the training and certification of individuals transporting and treating emergency traumatic and medical conditions enroute to the hospital or physician. In 1973, Congress passed a law authorizing the Department of Health, Education and Welfare to administer grants aimed at creating a uniform national emergency medical service system. Prior to that time, the Department of Transportation had been issuing some grants, aimed primarily at assisting needy or rural communities in obtaining ambulances. Several communities in Wyoming have obtained ambulances in the past under this program. One stipulation of the 1973 Emergency Medical Services Act, administered by HEW, directed each state to create an Emergency Medical Services (EMS) Department to receive, control and distribute grant funds. Such a department was already in existence in Wyoming under the auspices of the State Health Department and met the criteria outlined in the law.

Since 1972, the State Office of EMS has been operating a program to train personnel to render emergency medical aid. Approximately 3,000 Wyoming residents have completed the 80-hour basic Emergency Medical Technicians (EMT) course and are certified by the State Health Department as EMTs. Admittedly, many are not actively engaged in positions requiring their training and will be dropped from the Registry if they do not meet continuing medical education requirements for recertification every two years. However, those that do maintain the ever-increasing stringent requirements, will become an integral part of the developing statewide emergency medical system.

At the present time, in Wyoming, the emphasis on EMS development and expansion has been placed on a statewide communication system. One portion of emergency communications—the 911 telephone system—is now available in six of the seven major population areas within the state, and the seventh area will be completed this year. At the present time, the State of Wyoming Law Enforcement Communication System does not provide for direct interface between the 911 center and the hospital serving the center except in the five-county Big Horn Basin area. During the next year, an interface will be developed in the eight southern counties, between the 911 center and the hospital, utilizing a telephone line remote from the hospital local area and wide area systems, to the 911 center. The system in the Basin area is presently being utilized in two regional central medical emergency dispatch coordinating centers, and has proved extremely feasible and useful.

In addition to the local 911 center interface, it is proposed that regional centers be developed in Cheyenne and Rock Springs. Development of these centers will provide a basis for future regionalization of the EMS system in the Rocky Mountain Area. Regional centers will act as an interface point between Wyoming and the adjacent states. The center in Cheyenne will serve as an entry control point for Colorado and Nebraska, with the one in Rock Springs serving as an entry control point for Utah and western Colorado. Under this type of development, ambulances from the adjacent states will be able to call one central point to establish communications to notify the selected hospital that they are enroute. This interface will also serve as an aid in disaster and mass casualty situations occurring in the border areas

and in some of the less populated areas served by facilities of both Wyoming and those of neighboring states. Development of this interface with the states of Utah, Colorado and Nebraska was started last year and will proceed through 1977. It is proposed to develop the hospital paging capability in selected 911 centers in the less populated counties, and this will provide the capability to page ambulance attendants from the 911 center. Counties expected to utilize this system are Goshen, Platte, Carbon, Sweetwater and Albany. A similar system has already been developed in Laramie County. If Uinta County is included, the paging capability also would be extended to the Evanston 911 center.

The main emphasis, a statewide radio communications network between hospitals and ambulances, is in various stages of completion and development. It is currently in operation, utilizing private and foundation funds, in Campbell, Johnson, Big Horn, Park, Washakie, Hot Springs, Fremont and Teton Counties. The first HEW Emergency Medical Service Act grants under Title 1203 obtained by the State of Wyoming, are currently being utilized to develop a hospital-ambulance radio communications system in the eight southern counties of Goshen, Platte, Laramie, Albany, Natrona, Carbon, Sweetwater, and Uinta. This $350,000-portion of the network was completed in March 1977, and is now a functioning system of mountaintop transmitters, allowing ambulance to hospital radio communication in the sixteen counties mentioned. The next phase for the state EMS office is an application now pending, for an additional 1203 grant to create a similar network in the northeast counties of Sheridan, Crook, Weston, Niobrara and Converse. When fully integrated, the system configuration will also allow hospital-to-hospital communication if both utilize the same mountaintop repeater. It also allows hospital-to-hospital communication through two mountaintops via a UHF-VHF-UHF link. Ambulances in the area equipped with grant-supplied eight-channel VHF units will be able to use the various local and state emergency frequencies. It also allows for expanded crystallization to provide direct ambulance communication to law enforcement, or fire department, on high band VHF frequencies in operation and being developed by many local governments.

Another recent innovation in the statewide emergency medical system involves the use of either fixed-wing or helicopter evacuation of patients when the condition or distance to be traveled by ground ambulance is either undesirable or not feasible. The Armed Services have developed the Military Assistance to Safety and Traffic (MAST) program to aid civilian emergency medical services at both local and regional levels. Such a program was initiated at the F. E. Warren Air Force Base in Cheyenne in May, 1975. Helicopters, with military crews and medical equipment, respond to the following types of emergencies: serious medical emergencies resulting from highway accidents; inter-hospital transfer of patients with emergencies; premature infant transfers; the pickup and delivery of blood, medications and human organs for transplant; and, other emergencies from remote or inaccessible areas when ground transportation is not practical. The only disadvantage of the MAST program, in our statewide system, is its restriction for use to a 100-mile radius of Cheyenne.

Are we, as physicians, prepared to handle situations this overall emergency medical system creates? "The mere possession of a medical degree does not equip the physician to assume control of a mobile intensive care unit. Indeed, even advanced training and intra-hospital emergency and critical care medicine, or related specialties, rarely provides the necessary insight and capabilities to practice medicine by way of radio and telemetry. For this task, first-hand field experience is required, together with experience at the base station to provide a variety of unique skills and knowledge including the following: (1) Insight into the unique aspects of field conditions, particularly rescue, extrication, crowd control and lack of a priori physician authority at the scene; (2) Familiarity with radio communications and telemetry; (3) Ability to formulate protocols and standing orders; (4) Knowledge of health facilities in the region, particularly special care units, and their bed capacity and staffing at any given time; (5) Knowledge of the capabilities and limitations of the paramedics or EMTs, both in general, and, as applied to specific individuals; (6) Ability to teach allied health professionals; (7) Thorough familiarity with management of acute emergencies ranging from airway control and emergency cardiac care to emergency childbirth; (8) Ability to assess a patient when one cannot see, touch, auscultate or question, and (9) Ability to render treatment through the skills of another whose actions one cannot observe."[1]

Although there are no mobile intensive care units currently in use in the State of Wyoming, one can safely assume there will be in the future. Likewise, there are no advanced trained EMTs, or paramedics, operating here except in administrative positions. As mentioned previously, the State Office of EMS recently used 1203 grant funds to construct a radio communications system in the eight southern counties, and has applied for an additional grant for a similar network in the northeast counties. These 1203 funds are designated under the law as "basic life-support grants." Two other types of grants are also available. The 1202 funds, utilized as planning grants, have basically been bypassed here in Wyoming, up to the present time, because of adequate prior knowledge of our medical logistics. The 1204, or "advanced life-support grants," are available and it is anticipated the State EMS office will apply for this funding in the future. However, since this type of grant money will be used to purchase and equip mobile intensive care units, including cardiac telemetry, it will be delayed for lack of Wyoming hospital capability in this area. As allowed by recent changes in the Federal law, 1202, or planning grant money, can be utilized to evaluate the need and feasibility for 1204, or advanced life-support grants, in this state. This is currently under consideration.

Major changes in the State's emergency health care delivery system have occurred over the past several years, and further changes can be anticipated in the future. Many may question the need for the development of such a wide range and expensive system with all the problems it potentially produces for us as practicing physicians. However, like the Federal Interstate Highway System, we are but a small part of the overall national expansion. One might guess we are to live with the situation and accept it as a fact of life. As such, it behooves all of us to get "in on the ground floor," so to speak, to avoid the consequences of having a tiger by the tail. If our state physicians will become interested in participating and controlling the delivery of emergency medical services, the entire system could well become a benefit for us and our patients. As with many new ideas or practices, this could easily go beyond our control. One section of the EMS system development here in Wyoming requires the formation of emergency medical system councils in

each county. These councils are charged with determining the medical logistics and capabilities within their own area. If physicians would serve on these councils, we would be kept appraised of developments within our home counties. If we are involved with the training of the EMTs, we'll understand their capabilities and be able to rely on their judgment when called upon to handle emergencies via radio communication. Also, adequate control and standardization of the emergency health care delivery system will require, at times, regulatory legislation.

Summary

There have been major changes made, and planned, in the emergency medical care delivery system in Wyoming. As physicians, we may or may not be prepared for, or in favor of it. However, it would seem appropriate for us to continue our historical rule as "captains of the allied medicine ship" and get involved with the emergency medical system as it develops.●

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Changing trend of infant mortality

*Laramie County, Wyoming**

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There have been reports of dramatic decreases in the mortality rate of neonates who have received care in tertiary care, high risk nurseries.[1-4] During the past two years in Cheyenne, Wyoming, there has been a coordinated plan for the care of the newborn which has demonstrated a similar significant improvement and a striking change in neonatal mortality. The mortality rate of infants born in Laramie County, Wyoming, dropped from a level of twice that of Colorado and over fifty per cent more than the national average in 1971-1972 to almost one-third of that level by 1976. (Table 1.)

*From the Memorial Hospital, Cheyenne, Wyoming.

Materials and Methods

The neonatal service at Memorial Hospital in Cheyenne, a county hospital serving a population of approximately 80,000 people, was studied for the periods 1970-1971 and 1975-1976. (In 1970-1971 DePaul Hospital was included in the statistics for Laramie County, however, adequate statistics from F. E. Warren Air Force Base Hospital are not available.) Mortality data for all years included neonatal deaths which took place in Cheyenne as well as those that occurred after transfer to hospitals in Denver. (Table 2.)

TABLE 1

Neonatal death rates* in Cheyenne, Wyoming compared to State of Wyoming, State of Colorado and the United States

Cheyenne	20.3	25.3	14.7	7.8
Wyoming	15.0	15.3	N.A.	N.A.
Colorado	15.1	13.4	9.5	N.A.
United States	15.1	14.2	11.7	N.A.

*Deaths/1000 births

Results

Five factors were responsible for the decrease in the neonatal death rate. (Table 3): First, the newborn services in Cheyenne were consolidated. In the early 1970's there were three neonatal units — one at Memorial Hospital, a second at DePaul Hospital, and a third at the F. E. Warren Air Force Base Hospital. By 1972-1973 all three units were merged into an enlarged unit at Memorial Hospital. This consolidation converted three relatively small, inefficient nursery units in which outdated equipment was being used (because the small volume of babies in each separate hospital made it very expensive to purchase more up-to-date equipment) to a large, well equipped, adequately staffed newborn service for the care of normal and sick infants.

A second factor of importance was the increase in the number of deliveries performed by obstetricians. In the early 1970's obstetricians delivered approximately twenty-five per cent of the babies at Memorial and DePaul Hospitals in obstetrical units that did not contain fetal monitors. In the 1970-1971 period, the incidence of delivery by cesarean section, even when fetal distress was present, was relatively low. By the mid-1970's, obstetricians were performing more than eighty per cent of the deliveries, were utilizing fetal monitoring extensively, and were performing more and earlier cesarean sections for infants who exhibited evidence of difficulty. In 1975-1976 the cesarean section rate was fourteen per cent of all deliveries. The improved skill of obstetricians and the more modern technology available for the care of the feti and newborn infants resulted in infants who were less compromised at birth. Better prenatal care was reflected in lower perinatal mortality. (Table 2.)

TABLE 2

Newborn statistics for Cheyenne, Wyoming

	1970*	1971*	1975 +	1976 +
Births in Cheyenne	837	788	1091	1024
Deaths in Cheyenne	17	20	12	5
Transfers to Denver	2	none	21	28
Transfer deaths in Denver	none	none	4	3
Transfer survivals	100%	- - -	81%	93%
Total Cheyenne Neonatal deaths	17	20	16	8
Cheyenne neonatal deaths/1000 births	20.3	25.3	14.7	7.8
Cheyenne stillborns	4	11	10	8
Perinatal deaths/1000 in Cheyenne	25.1	39.3	27.5	15.6
Delivered by Cheyenne obstetricians	175	210n	891	846
Percent by Cheyenne obstetricians	21%	26%	81%	83%

*Combined Memorial and De Paul Hospitals
+Memorial Hospital
In 1972 the De Paul unit merged with Memorial.

A third factor which influenced the decrease in neonatal mortality was the addition of pediatricians to the community. No pediatricians were in practice in the area in the early 1970's. By the mid-1970's, there were three and a fourth began to practice during 1976. The pediatricians had received thorough training in neonatal care and had established a good working relationship with the high risk newborn centers to which they referred patients (i.e., Colorado General Hospital, Denver Children's Hospital, and Fitzsimons Army Hospital.) With the advent of improved pediatric care for newborn infants, many additional pertinent laboratory studies were carried out including data obtained from umbilical artery catheterizations, blood gas determinations, special roentgenographic studies, cultures, blood chemistries, etc., which were used to distinguish between the seriously ill infants who required specialized care and those sick infants who could be satisfactorily treated in the hospital where they had been born. Criteria were established which helped identify most infants who were at high risk for complications and would probably require ventilatory assistance

TABLE 3

1. Consolidation of small inefficient, out-dated newborn units.
2. Modern obstetrical care - - prenatally and at birth.
3. Modern pediatric care of the newborn.
4. Utilization of an experienced and well supervised transport team with modern equipment.
5. Utilization of tertiary care centers for high risk, sick newborns.

or special procedures requiring tertiary care. Thus transportation was arranged before those babies became critically ill and could not be moved. High risk infants were transferred with greater frequency in the 1975-1976 period but not until their condition had been stabilized at Memorial Hospital by the pediatricians and the transport team. Moderately sick and low risk infants remained in Cheyenne for care and treatment (i.e. infants with hypoglycemia, suspected sepsis, moderate respiratory distress, feeding problems, etc.)

The fourth factor of importance in decreasing neonatal mortality was the transfer of seriously ill patients to regional, tertiary care nurseries. The transfers were made by an air transport service utilizing well trained and competent nurses and an inhalation therapist with expertise in the use of portable infant ventilators. Stabilization in the condition of infants requiring transfer before they were sent to the intensive care centers contributed to the decrease in the mortality rate.

Finally, the level of care now being provided in tertiary care centers has dramatically improved the outcome of small, preterm infants and sick infants. Most of the babies transferred to Denver required respirator therapy for days to weeks yet their survival rate was eighty-one per cent in 1975 and ninety-three per cent in 1976! The presence of full time neonatalologists, well trained and experienced nursery nurses, and the variety of sophisticated equipment provided a level of care heretofore not available to high risk newborns.

Summary

The Laramie County experience demonstrates that an appreciable improvement in the neonatal mortality rate can be obtained by consolidation of nursery services, utilization of modern obstetrical care and modern pediatric care for the newborn, and skilled transfer of very sick infants to centers where there are personnel and equipment to care for difficult problems. It is recommended that inefficient nursery units with few babies, inadequate personnel, and out of date equipment be phased out and consolidated into larger and better equipped and staffed regional units.

Obstetricians and pediatricians should have a more active role in providing maternal and newborn care, particularly in high risk pregnancies or where problems exist or could occur.[5] All health professionals who are responsible for deliveries must update their technical skills so they can use and interpret antenatal fetal monitoring and other supportive equipment; know how to perform umbilical arterial catheterization; and have blood gas analyses available; be able to interpret blood gas data, pertinent blood chemistries, and diagnostic roentgenograms. Severely sick infants should be transferred to a tertiary level nursery staffed by full time neonatalogists.

The institution of such a coordinated program will result in a significant improvement in morbidity and mortality even in relatively small communities considerable distances from medical centers with tertiary level nurseries. The data from Laramie County, Wyoming, would support such an effort. •

ACKNOWLEDGEMENT

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Gram negative bacilli in sputum

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It has been estimated that five-tenths to five per cent of all patients admitted to a hospital develop nosocomial or hospital acquired respiratory infections. Gram negative organisms have been responsible for an increasing per cent of these infections in recent years.

It is not uncommon for the physician to be confronted with a sick patient with a fever. He naturally looks for the common clues in the history and physical. Often these patients have a cough and sputum or frequently secretions from a tracheostomy tube. One of the pieces of data he may obtain is that the saliva, sputum, or tracheal secretions show gram negative bacilli by gram stain or culture. Notwithstanding other sources of infection, how is this information interpreted? Should it be treated or ignored? We shall try to place this finding in perspective.

First, gram negative rods are likely to be found in the pharynx of normal persons. Rosenthal has shown that about thirty per cent of normal people harbor gram negative organisms in their posterior pharynx.[2] Eighteen per cent of these gram negative rods were considered potentially pathogenic, i.e.: Enterobacteriaceae and Psuedomonas aeruginosa. This study of one hundred persons excluded those with chronic obstructive pulmonary disease, ear infections, respiratory illness within four weeks, hospitalization or antibiotics within four weeks, medical personnel and persons with acute illness.

It has also been demonstrated that the sicker a patient is, the more likely he is to harbor gram negative organisms. Johanson studied persons in stages of health ranging from normal to moribund, and found an increasing incidence of gram negative bacilli with increasing degrees of illness.[3] Normal subjects in this study had a six per cent incidence of gram negative cultures, psychiatry patients in the hospital and otherwise physically well had six per cent, moderately ill patients had thirty-five per cent, and moribund patients likely to be found in intensive care units had seventy-five per cent incidence of gram negative rods. Exogenous exposure to inhalation equipment and antibiotics could not be shown to account entirely for this difference in this study, although it has been verified that liberal use of penicillin and other antibiotics active against gram positive organisms will select for gram negative rods.[4] Also inhalation equipment has been incriminated as a source of gram negative organisms.[5] So, just the presence of a gram negative bacillus in the sputum does not mean infection. It may only represent colonization.

How does one decide, then, whom to treat? There are many clues but even then the decision can be difficult. The history may indicate chest pain, shortness of breath or shaking chills. When the sputum is green, yellow, bloody or brownish in color, it is more likely infected. If it has a foul smell the chances of infection are even greater (think of anaerobes.) Physical examination may identify the rough breath sounds of tracheitis, wheezes of bronchitis, the rales of pneumonia, or the dullness of empyema. A chest x-ray may show an obvious infiltrate or pleural effusion.

Transtracheal aspirate can be helpful, since all respiratory secretions below the cricothyroid membrane should be sterile. There are a few tricks, however, if one must rely on expectorated sputum. The patient can gargle several times with tap water to clear loose contaminating secretions in the mouth, and gram stain of the sputum should then show a better correlation with actual flora below the cricothyroid membrane.[6] Organisms in association with granulocytes are more likely to be pathogenic

than if associated with squamous epithelial cells.

If, by these criteria, one decides he is dealing with a gram negative infection, treatment with antibiotics most likely to be effective should be started according to known sensitivity studies of the predominant gram negative organisms in the hospital while awaiting culture and sensitivity data. Periodic gram stains and culture of sputa of sick patients can sometimes give the physicians a big jump on treatment of these infections.●

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Post partum nephrosclerosis

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A syndrome of irreversible renal failure in the post partum period is being recognized with increased frequency. This entity has been described under several synonyms: post partum hemolytic uremic syndrome, irreversible post partum renal failure and post partum nephrosclerosos.[1–11] The etiology is unclear but the changes in the kidney, consisting histologically of fibrin deposition and thrombi within the glomeruli and vasculature, result in characteristic changes of nephrosclerosis and ischemic glomeruli.

Early recognition is desirable because of the possible reversal of the syndrome with anticoagulant therapy.[6 - 11] The common mode of clinical presentation has been hypertension with encephalopathy, uremia, or in some cases, hemorrhage secondary to diffuse intravascular coagulation. A universally grave prognosis has been reported even with chronic dialysis therapy.[10] We report three cases of post partum nephrosclerosis, each of whom presented with far advanced uremia and hypertension in post partum followup. All three cases were successfully managed by hemodialysis and transplantation.

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CASE REPORTS

Case 1: A 29-year-old Caucasian woman with history of mild hypertension developed severe hypertension in her first pregnancy at 20 weeks gestation. Fetal demise occurred and C-section was performed. Moderate doses of antihypertensives were required. She was discharged from Irwin Army Hospital, Fort Riley, Kansas, normotensive and with a normal creatinine clearance. During the next two weeks she developed edema, shortness of breath, nausea,

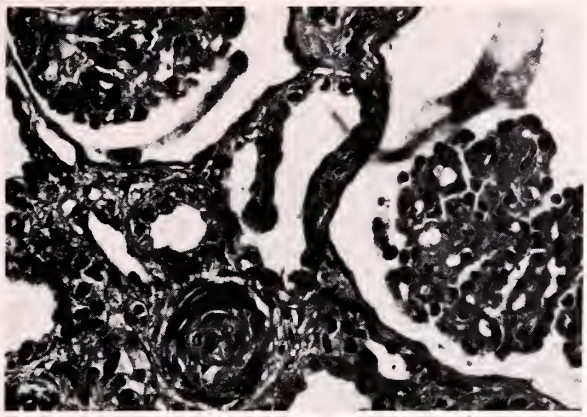

Fig. 1. Biopsy from Case 1 showing intimal proliferation of arteriole and thrombosis with muscular hypertrophy. Glomeruli are shrunken and ischemic with areas of necrosis (Hematoxylin and eosin X 325).

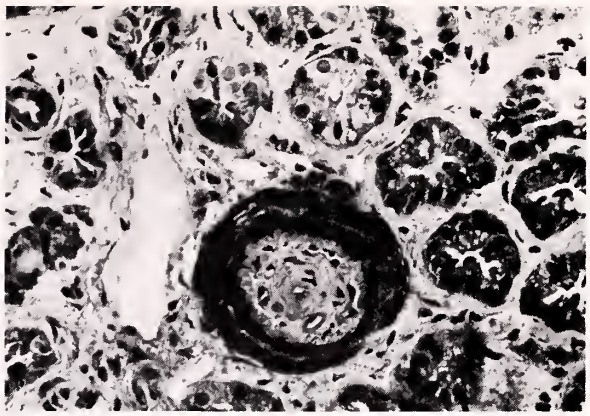

Fig. 2. Typical arteriole from Case 2 showing intimal proliferation and fibrosis, total occlusion from thrombosis and thickened muscular wall. (Masson trichrome X 375).

vomiting, weakness and oliguria. She presented with severe azotemia at readmission and was transferred to Fitzsimons Army Medical Center on July 30, 1973.

Physical examination on admission showed blood pressure of 210/125 with papilledema and retinal hemorrhages. A pericardial friction rub was present with a third heart sound and edema. BUN was 108 mg% and serum creatinine 22 mg%. Urinalysis showed WBC's, RBC's and protein. The chest x-ray showed pulmonary edema with bilateral pleural effusions, cardiomegaly and interstitial infiltrates. An IVP failed to visualize and retrograde pyelogram revealed no obstruction. There was no evidence of microangiopathic hemolytic anemia or DIC. Platelet count and fibrinogen levels were normal.

The patient was hemodialyzed. An open renal biopsy revealed severe arteriolar nephrosclerosis with ischemic glomerular changes (Fig. 1). Heparin therapy was instituted one week later but resulted in a severe retroperitoneal hemmorrhage resolving after cessation of heparin. Bilateral nephrectomy was performed October 10, 1973, to control blood pressure. On renal dialysis the blood pressure remained high until six weeks later when she finally attained normotension. She subsequently received a successful cadaveric renal transplant on November 29, 1974 and continues to have normal renal function.

Case 2: A 20-year-old Caucasian woman was in excellent previous health with no history of hypertension. She gave birth to her second child on December 28, 1974, without complications. Two weeks post-delivery, she developed vaginal bleeding and her hematocrit fell from 40 to 20. Two weeks later her blood pressure was normal but protein was noted in the urine. She later developed nausea, vomiting, and persistent vaginal bleeding. On January 27, 1975, she was hospitalized and at D & C small amounts of retained placental fragments were found. The BUN on admission was over 100 mg%, the serum creatinine 23 mg% and potassium 5.8 mEq/L. The platelet count, fibrinogen, PT and PTT were within normal limits. She developed shortness of breath, hypoxia, and oliguria, and underwent hemodialysis at Landstuhl Army Hospital. She was transferred to Fitzsimons Army Medical Center on February 20, 1975.

Physical exam showed a blood pressure of 160/90, normal funduscopy and a Grade II systolic ejection murmur. Pelvic examination was within normal limits. She remained oliguric and severely azotemic. There was no evidence of microangiopathic hemolytic anemia, DIC or TTP. Retrograde pyelogram showed no obstruction and an open renal biopsy demonstrated severe arteriolar nephrosclerosis (Fig. 2). Despite ultrafiltration on dialysis, the patient became severely hypertensive and developed encephalopathy with retinal hemorrhages. Therapy with Minoxidil and Inderal was instituted with complete control of her hypertension after standard antihypertensive therapy was ineffective. She is currently being evaluated for transplantation and remains asymptomatic.

Case 3: A 32-year-old black woman in the 34th week of her first pregnancy developed severe headaches, edema, and proteinuria. Her blood pressure was 180/120. She was treated with diuretics, magnesium sulfate, phenobarbital and apresoline with poor response, and a C-section was done on April 3, 1970, with delivery of a normal infant. She continued to be severely hypertensive postoperatively. The BUN and creatinine were normal preoperatively, but despite good urine volumes, she developed increasing azotemia postoperatively. On the 16th postoperative day the BUN was 120 mg% and the serum creatinine 10 mg%. She underwent peritoneal dialysis and was transferred to Walter Reed Army Medical Center on May 20, 1970. There she remained severely hypertensive with encephalopathy, hemorrhages, and exudates in both fundi. Despite ultrafiltration on hemodialysis, hypertension persisted. Over the next two months, however, this spontaneously improved and she subsequently did not require antihypertensives. A renal biopsy on July 21, 1970 showed severe nephrosclerosis and ischemic glomerular changes. She underwent cadaveric transplantation in October 1973, but this was followed by accelerated graft rejection, and she is now doing well on three times weekly hemodialysis.

Discussion

Thirty-two cases of irreversible renal failure in the post partum period have been reported. A spectrum of renal disease exists, as some cases have recovered spontaneously and the renal involvement was only minimal, detected by biopsy.[10] The more severe cases, even when treated with hemodialysis, have had a universally bad prognosis.[9] Our three cases have had long term survival with treatment by hemodialysis and/or transplantation.

The etiology of this syndrome remains unclear. Ergot derivatives, vasopressin and birth

control pills have been implicated but not proven.[2] Pregnancy, of course, is a predisposing factor. The histologic picture is quite characteristic but not pathognomic.[1] The most typical lesion is the renal vascular involvement consisting of intimal fibrosis obliterating the lumina of blood vessels. Fibrin deposition and fibrin thrombi are frequently found within the glomeruli and vasculature of the kidney. The glomeruli are shrunken and appear ischemic. All three of our cases showed the typical histologic lesions described in the literature.

Most reported cases have been associated with microangiopathic hemolytic anemia. None of our three cases had evidence of hemolysis. One patient did have vaginal bleeding early post partum which may have indicated a bleeding diathesis but this may have been attributable to uremia. It is possible that these individuals went through a phase of hemolytic anemia and thrombocytopenia before they arrived at our hospitals.

These patients generally present from two to six weeks post partum with uremia, hypertension and anemia. Specifically, nausea, vomiting, shortness of breath, edema, oliguria, encephalopathy or bleeding disorder may bring them to medical attention. The hypertension has been a severe problem in our patients, with encephalopathy present in all at one time or another. Bilateral nephrectomy may be required to control hypertension[5], but this is only after an aggressive trial of antihypertensive therapy, including the new oral vasodilator, Minoxidil. Early recognition of this syndrome is of importance since there is some evidence that anti-coagulant therapy may reverse this disorder in the early phases.[6,11] A trial of heparin should be attempted. However, if renal biopsy is contemplated, the anticoagulants should be stopped first.

Previously this disorder has been associated with a universally bad prognosis despite hemodialysis. In our experience, aggressive dialytic therapy and/or transplantation is successful. One of our three patients has received a successful cadaveric transplant and is surviving with normal renal function two years later. Another received a cadaveric transplant but had an acute rejection unrelated to the underlying disease. All three patients received hemodialysis successfully and two of them are still surviving on dialysis two and four years later.

Summary

Post partum nephrosclerosis may result in irreversible renal failure following an otherwise uncomplicated pregnancy with progressive azotemia and hypertension occurring in the first six weeks post partum. Three patients who survived this syndrome are presented, emphasizing the clinical recognition and the successful treatment by hemodialysis and renal transplantation.●

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Value of knee arthroscopy*

Charles D. Magill, MD, Denver

Arthroscopy has become available in most areas as more and more surgeons learn the technic. The purpose of this review is to describe the indications and list the likely results of arthroscopy of the knee. This review is directed at all physicians who wish to know when this examination is advisable.

The procedure itself is simple. It is performed on an outpatient basis under local or general anesthesia. It is performed through a stab incision. A typical examination might include the following: (a) manipulative examination under anesthesia, (b) joint aspiration, (c) thorough irrigation of the joint, (d) biopsy, (e) photographs, and (f) other therapeutic procedures. Biopsy reports, operative records, and photographs become an indispensable part of the patient's chart. The patient returns to work, school or sports within forty-eight hours without interference of knee function.

Indications

Arthroscopy of the knee cannot replace clinical examination and routine radiographs as the initial diagnostic procedure in evaluating the knee. Next, arthroscopy must be considered because it can provide a more accurate diagnosis than available through other means. Indications for arthroscopy are listed in Table 1, the usefulness of each being graded on a scale of 1 to 4.

The adolescent knee with persistent symptoms but minimal findings, including negative arthrograms, puzzles every physician. Arthroscopy refutes or supports the diagnosis; abnormalities can be directly visualized through the scope. In addition, the procedure frequently relieves symptoms indefinitely and the small stab incision is cosmetically acceptable. Empiric amelioration of symptoms occurs in about sixty per cent of examinations regardless of the underlying cause.[3]

Arthroscopy is indicated when the arthrogram is ambiguous or negative. Structures seen by arthroscopy but not by arthrography include (a) type of snyovial fronds, (b) appearance of the articular cartilages, (c) condition of the anterior cruciate ligament, (d) the extent of synovitis, (e) the location of a cartilaginous loose body, and (f) the direct visualization of the menisci. However, if arthroscopy is not available, certainly arthrography must be considered. Both procedures are more bearable than "exploratory arthrotomy." According to Jackson[3], clinical examination of the knee is accurate only about seventy-five per cent of the time, with reliable arthrography raising this figure to ninety per cent. With arthroscopy, a diagnostic accuracy of ninety-eight per cent is achieved. However, this degree of reliability can only be achieved by experience with several hundred patients.[1]

Should the knee with clear-cut signs and symptoms still have arthroscopy? A knee with an obvious tear of the meniscus has been found to have a tear of the opposite meniscus in five to twenty per cent of patients.[2] Arthroscopy provides a better view of the lateral meniscus than arthrotomy

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through a medial incision. In addition, in about half of the cases where a tear of the meniscus was suspected, arthroscopy either failed to detect the tear or instead discovered a totally different problem. Such problems were (a) tear of the opposite meniscus, (b) chondromalacia of the patella, (c) isolated tear of the anterior cruciate ligament, (d) other lesions, and (e) nothing at all. Meniscectomy would have added an unnecessary hazard and morbidity to these patients.

The knee with obvious arthritis in need of surgery can benefit from preoperative arthroscopy. The extent and type of pathology can help select the proper procedure, whether it be debridement, osteotomy, replacement, or arthrodesis. Incisions can be better planned. Some patients will have relief of symptoms which might postpone surgery.

Should arthroscopy be performed on every knee? The common cold provides a good analogy. Should a chest radiograph be obtained in every case of the common cold? Certainly, many knee problems, like many common colds, will do well with simple treatment and observation. However, when doubt arises, when the patient is not doing well, when there is a conflict of information, or when simple treatment is not enough, arthroscopic consultation is advisable.

Therapeutic Procedures with Arthroscopy

Miniaturization of tools and development of new instruments will make arthroscopic surgery more practical for many procedures now done by open surgery. Even now, however, some benefits have occurred. Crystals which may have accumulated for years can be washed out with immediate and lasting benefit.[4] Rupture of small adhesions can result in improved motion and relief. Biopsy of a suspicious nodule under direct vision can yield better results than with blind technics.

CASE REPORT

A 21-year old man sustained a puncture wound to the suprapatellar area of his right knee. A roofing staple coated with plastic had penetrated his clothing, soft tissues, and anterior femur. He pulled the staple out leaving behind the plastic fragments. Arthroscopy was performed, the wound was inspected, the plastic was washed out by thorough irrigation and the wound was irrigated with neomycin, bacitracin, and polymyxin B. Systemic antibiotics were given. He was discharged the following day. He had no further problems on postoperative followup.

TABLE 1

Indications	Usefulness
Adolescent Knee	++++
Arthrotomy doing poorly	++++
Litigation	++++
Negative arthrogram	++++
Inconsistent arthrogram	++++
Synovial biopsy	++++
Psuedogout	++++
Disability determination	++++
Persistent effusion	++++
Select proper reconstruction	+++
Make smaller incision	+++
More rapid diagnosis	++
Obvious tear of meniscus	++
Treatment of defenerative arthritis	++
Foreign body	++
Loose body	++
Patellar subluxation	++
Contracture	+
Hemarthrosis	+
Pyogenic infection	+

*Scale defined in text.

Results of Arthroscopy

Direct visualization of intraarticular structures means a high rate of diagnosis. Exploratory arthrotomy, removal of a normal meniscus, large serpentine incisions, and prolonged unnecessary observation should become things of the past. Immediate benefits include avoiding an open operation, performing the procedure through the arthroscope, executing a more precise operation, and empiric improvement of symptoms. Other benefits include better evaluation of long-term followups and better understanding of knee disorders.

Complications

Arthroscopy is a technic which demands experience and practice. Most errors occur in the first 100 cases, as one learns the technic. An error rate of two per cent is expected in experienced hands.

General or spinal anesthesia permits use of the larger, operating, wide-angled arthroscopes as well as complete relaxation of the knee. Arthroscopy as an isolated procedure has produced no reported instances of mortality, infection, thromboembolism, or joint

TABLE 2

Therapeutic Procedures with Arthroscope

Procedure	Usefulness*
Irrigation of joint, routine	++++
Biopsy	++++
Rupture small adhesions	++++
Irrigation for gout, pseudogout	++++
Loose body removal	+++
Bucket handle tear meniscus remove	++
Divide constricting plica	++
Remove small fracture fragment	++
Remove foreign body	++
Install drainage tubes	+
Antibiotic irrigation	+
Improve motion in stiff knee	+
Remove hemarthrosis	+

*Scale defined in text.

damage. Breakage of the newer fiberoptic arthroscopes inside the joint has not occurred. Postoperative effusion and stab-wound irritation have occasionally occurred. Interference of knee function or aggravation of the knee problem has not occurred.

Summary

A brief review of the current role of arthroscopy of the knee is provided so that physicians may know when this examination is advisable. The procedure is easily tolerated by patients. Many patients are empirically improved. Unnecessary knee operations are avoided. Necessary surgery is better guided. The diagnostic accuracy is ninety-eight per cent in experienced hands. A short case report demonstrates surgery through the arthroscope.

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Van Tassel House near Tarrytown, the residence of Washington Irving.

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Misdiagnosis of fetal heart rate

*An unusual case of mistaken identity**

Richard P. Perkins, MD, and Michael R. Soules, MD, Denver

A case of supposed fetal death, with suspected trans-fetal recording of the maternal pulse is presented. Similarities between fetal and maternal pulses, plus failure to observe or initiate differentiating clues led to the misdiagnosis.

An occasional trap when observing electronic fetal heart rate monitoring tracings obtained through a fetal scalp electrode is that of recording the maternal pulse transmitted through an undiagnosed dead fetus. Reports concerning this possible complication have appeared in the past.[1] The following case report illustrates the remarkable story of a patient and her fetus in whom the opposite error was made. Clinical circumstances and observations led to the misdiagnosis of fetal death with recording of the maternal pulse when in fact the fetus was inexplicably still alive. The extraordinary management of the patient for the purpose of avoiding cesarean section for a dead baby resulted in the delivery of a depressed but surviving infant - a condition which we have called the "Lazarus syndrome", for our own reference in the future.

CASE REPORT

An 18 year old, gravida 2, para 0, surgical abortion 1, was admitted to the hospital at 39 weeks' gestation. She had been followed in the Outpatient Clinic since 28 weeks' gestation with fundal McDonald measurements slightly greater than dates for the first four visits. Her baseline blood pressures had averaged 120/75 mmHg. There had been no complications noted. One week prior to admission, the patient was seen with a blood pressure of 120/84 mmHg. and no proteinuria. She had been advised to maintain bedrest at home. On the day prior to admission, the patient was seen with a blood pressure of 140/94 mmHg. which was later repeated and found to be 136/100 mmHg. There was no proteinuria. She was advised to maintain bedrest at home and be seen in the Clinic in three days. On the morning of the following day, the patient awoke with a severe headache. Her family observed that she was "bumping into things" when up and about. Shortly thereafter, she began to have major motor seizures and, within a 45-minute period at home, sustained five such episodes without regaining consciousness.

The patient was brought to the Emergency Room where she was given a slow continuous dose of 5 gm. of magnesium sulfate intravenously and transferred to labor and delivery. Her blood pressure in the ambulance had been recorded at 190/100 mmHg. On admission to labor and delivery, it varied from 164/102 mmHg. to 200/100 mmHg. Initial auscultation of the fetal heart showed a heart rate of 110 beats per minute increasing to 120. The early endeavors were directed at correction of maternal acidosis (arterial pH-7.15, pO_2-93, pCO_2-23). Pelvic examination revealed the cervix to be 1-2 cm. dilated, 90% effaced and the vertex was at -1 station. Estimated fetal weight was 3,000 gms. The patient had no further convulsions following her arrival in the Emergency Room, but was hyperactive and disoriented requiring soft restraints and sedation with intravenous barbiturate in addition to magnesium sulfate.

Following stabilization, repeat arterial blood gases were pH-7.40, pO_2-121, pCO_2-greater than 25. Potassium was 2.8 mEq/L. BUN was 6 mg./dL., creatinine 1.4 mg./dL. Blood pressure ranged around 180/120 mmHg, and a catheterized urine revealed 2+ proteinuria. Intravenous controlled infusions of magnesium sulfate, Apresoline®, and eventually oxytocin were begun to control the patient's status and to increase labor. External fetal monitoring disclosed a steady heart rate in the range of 150 beats per minute. At the onset of induction, the membranes were ruptured and an internal electrode applied with an internal catheter for recording uterine pressures (Fig. 1). The amniotic fluid at rupture of membranes was thick and bloody, having the appearance of a recent fetal demise. The examiner was strongly suspicious that the fetus was dead, and attention was turned to the heart rate tracing obtained. This was found to correspond closely with the maternal pulse, at a rate of 140-150 beats per minute. There was almost complete absence of baseline variability. The electrode was repeatedly checked and found to be on the fetal presenting part without unusual contact with maternal tissues. It was then decided that the fetus was dead and that every effort would be made toward vaginal delivery.

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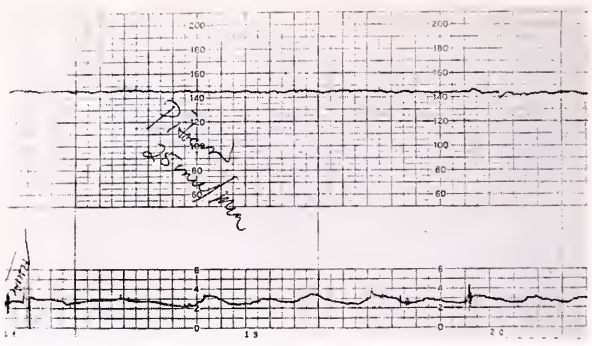

Fig. (1.) Internal cardiotocography, with pressure catheter functioning. Note rate of oxytocin infused at 25 mU/min. and poor contractile pattern with elevated resting tone. Fetal heart rate tracing shows minimal to absent baseline variability.

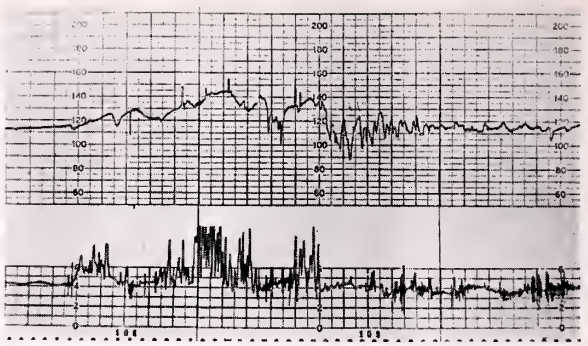

Fig. (2.) Late stage of labor. Patient vomiting and pushing. Note acceleration of the heart rate recording which would have been atypical if maternal heart recorded. Note also a brief burst of increased baseline variability suggestive of fetal arousal from depressed state by preceeding events.

Comparatively little attention was subsequently paid to the fetal heart rate tracing in light of the reasonable conclusion that the baby was not alive. The usual availability of fetal EKG monitoring tracings on one of our machine was at the time not functioning. Considerable difficulty was encountered with regulation of uterine activity with the intravenous oxytocin infusion and therefore a markedly prolonged, hypertonic labor ensued (Fig. 2). Ultimately, the patient became fully dilated with the head not completely engaged; with the combination of the Malmstrom extractor and the Barton forceps for a deep transverse arrest, the patient was delivered with some difficulty and moderate maternal trauma. To the surprise of the physicians in attendance, the infant was a 4140 gm. male with early signs of life. The Apgar score was 3 at one minute and 6 at five minutes after institution of vigorous resuscitative efforts. No evidence of placental abruption was present. The baby's subsequent course was complicated early by lethargy and hypotonia, progressing later to hypertonia with ultimate resolution. At two weeks of age there were no obvious neurologic or performance abnormalities.

After repair of maternal, vaginal, and perineal lacerations associated with delivery, the mother pursued a febrile course despite antibiotic administration. Late in her hospital course, a 4th-degree laceration acquired at delivery broke down with drainage of some purulent material and resolution of her persistent fever. The patient had been heparinized for chronic tachycardia and pyrexia because of the suspicion of an associated pelvic thrombophlebitis. Heparin was continued through her discharge. The mother was discharged afebrile on the 14th hospital day. The baby was discharged with her.

Discussion

In light of the recognized potential for recording maternal pulse through the fetal scalp in cases of a dead fetus, and because of the overwhelming suggestions of fetal demise in the present severe clinical circumstances, the initial impression, based on the similarity between fetal and maternal pulses and the lack of customary baseline variability in the fetus led to the understandable conclusion that the baby was not alive. Several examiners had participated in the relevant observations and arrived at the same conclusion. It seemed to those in attendance incredible, with the great difficulty in achieving good intercontractional uterine relaxation with repeated attempts at standardization and surveillance of the intrauterine catheter, that the fetus could possibly be alive.

Several additional methods for determining fetal life or distinguishing fetal cardiac activity from that of the mother could have been attempted. Had any reasonable suspicion of circumstances other than those presumed existed, undoubtedly some or all of the following methods would have been tried:

(1) Attempts at reduction of maternal heart rate: Certain maneuvers could have been tried to produce diagnostic changes in maternal heart rate. Carotid sinus massage, if successful, should have produced a noticeable decline in maternal heart rate, even with the coincident administration of hydralazine. Should no coincident drop in the recorded heart tones have occurred, the assumption could have been made of fetal life. Alternatively, the intravenous administration of atropine might have produced a diagnostic rise in maternal heart rate. This would have had to have been carefully documented as to time of onset in relation to the fetal heart rate tracing, since atropine does cross the placenta and might be expected to create a similar rise in fetal heart rate. Had the patient not been hypertensive, small amounts of intravenous sympathomimetic drugs (e.g., phenylephrine) or of propranol might also have been used. In this case, the patient's hypertension and poor perfusion would have theoretically precluded the use of either of these drugs. Again, the necessity to record the effects on the maternal heart rate as temporally related to those of the "fetal" heart rate would have been necessary.

(2) Fetal EKG: Some electronic fetal monitoring equipment has the capability of direct recording of the fetal electrocardiogram. Had this been accomplished and compared with the rate and wave form of the maternal EKG, a differentiation might have been possible.[2]

(3) Fetal scalp blood sampling: An attempt at obtaining fetal scalp blood for pH determinations might have been useful. The expectation would have been for an extremely low pH in this circumstance on admission, especially in light of the maternal pH of 7.15. However, with a dead fetus the expectation would have been either failure to obtain any blood for analysis, or the finding of a sample with pH substantially less than 7.00. Anything above that, properly obtained, would have mitigated against the existence of fetal death.

(4) Doppler fetal heart tone assessment: The standard Doppler principle fetal heart tone monitors have a distinctive biphasic sound when over the fetal heart, as opposed to all other maternal blood flow sounds recordable from the pelvis. Simple auscultation in this patient was not possible because of her noisy and physically uncontrollable behavior. In this case, as opposed to most others, the simpler machine would have given correct information, while the use of the more complex apparatus resulted in an inaccurate conclusion.

Considering the comparatively minor degrees of maternal vascular disease and other causes which give rise to intrapartum fetal death, the fact of survival of this infant under these circumstances is almost beyond belief. The apparent survival of the infant without demonstrable neurologic abnormalities is even more incredible. That an infant in this severe climate of maternal vascular disease could tolerate persistent uterine hypertonus and sustained hypoxia in labor challenges comprehension. Taking all considerations into account, the conclusions of the physicians responsible for determination of maternal and fetal welfare in this case are entirely understandable. The case serves as a testimonial to the capacity of this fetus to withstand enormous insult and to survive apparently unharmed. The ultimate status of the child will bear close watching.

The case also serves to illustrate how being prepared not to be trapped by electronic fetal heart rate monitoring artifact of one type can predispose to another erroneous conclusion. Had the involved physicians been less well prepared against being deceived, they might have acted in the interests of the fetus on the assumption that what was recorded was in fact what was expected to be recorded. It is hoped that the above "tricks" designed to differentiate maternal from fetal heart rate recordings will serve to further enlighten those who have the misfortune to be faced with a similar dilemma in their own experience.●

REFERENCES

[1] Paul, R.H. Petrie, R.H.: Fetal Intensive Care: Current Concepts. *A publication of the University of Southern California School of Medicine, Post-graduate Division;* 1973, pg. 64.

[2] Figueroa-Longo, J.G., Poseiro, J.J. Alvarez, L.O., Caldeyro-Barcia, R.: Fetal electrocardiogram in term labor obtained with subcutaneous fetal electrodes. *Am J Obstet Gynecol* 96:556, 1966.

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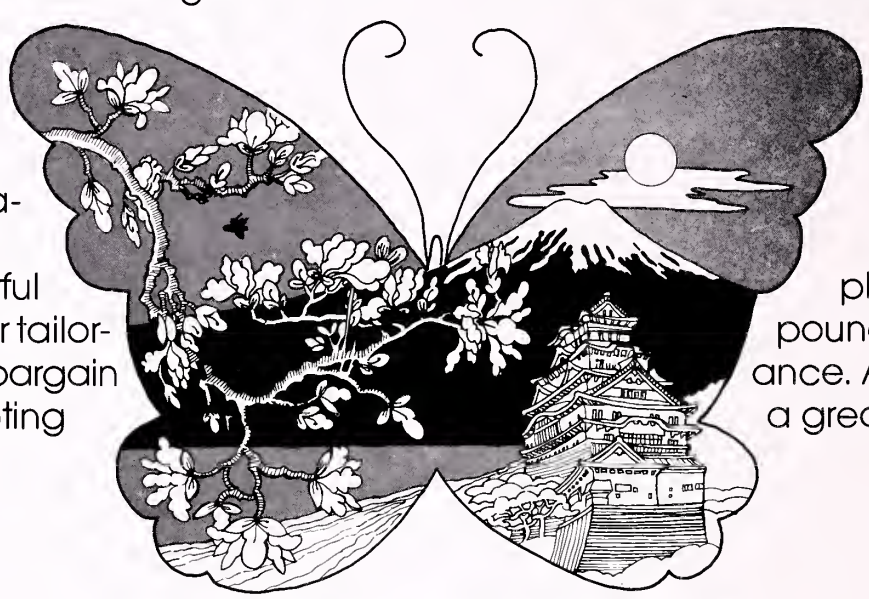

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WYOMING

Wyoming Plans for Continuing Medical Education

The Continuing Education Committee of the Wyoming State Medical Society and the developing College of Human Medicine at the University of Wyoming will jointly plan and implement a state-wide program of continuing medical education. This joint effort began in May when John Corbett, MD, Chairman of the Wyoming State Medical Society's Committee on Medical Education appointed David M. Holden, MD, Assistant Dean for Clinical Sciences at the College of Human Medicine in Laramie to be Chairman of the Continuing Education Committee of the Wyoming State Medical Society.

On June 10, 1977, a joint meeting of the Continuing Medical Education Committee of the Wyoming State Medical Society and the staff of the College of Human Medicine was held in Casper. The Wyoming Hospital Association was also represented.

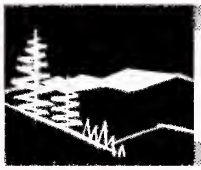
The Continuing Education Committee's workshop identified several areas of immediate need. The Committee agreed that a needs assessment including a questionnaire to determine the current continuing education efforts and needs of practicing Wyoming physicians should be designed. It was felt that a calendar of Continuing Education events throughout the State of Wyoming should be developed and maintained to assist Wyoming physicians. This calendar would describe CME programs by specialty and by disease content. If this were done a physician could call and ask "what's going on in diabetes?" and receive an answer to where and when the next program that answered this need was planned within the state. A record keeping system of continuing education credits and an ongoing evaluation of CME programs was also envisioned.

An important planning dimension was the provision for regionalization of continuing education efforts. It was felt that in the large geographic area of Wyoming, a state-wide program would not be as effective as one which was directed centrally, but which was managed through seven regional coordinators. The planning process will be continued by seven regional representatives who will determine the criteria for selection and desirable qualifications for regional continuing education coordinators. The planning group will finalize a survey questionnaire to be sent to all Wyoming practicing physicians and will come up with firm recommendations for the appointment of permanent regional continuing education coordinators who will also need funding for travel, program development and staff support. This planning group will meet in mid-July, and a report to the full Continuing Education Committee will be made in conjunction with the Annual Meeting of the Wyoming State Medical Society meeting in August.

Dr. David Holden, Chairman of the Wyoming State Medical Society's Committee on Continuing Education feels that funding for continuing education will be sought from a number of sources including the College of Human Medicine, the Wyoming State Medical Society, from its membership, and from foundations.

At this full day workshop meeting, Dean Robert M. Daugherty of the College of Human Medicine pointed out the linkage of undergraduate and residency programs with continuing professional education. He said, "Continuing Medical Education is a top priority of the College of Human Medicine for several reasons. First, we feel that the opportunity to obtain quality continuing medical education experiences in medium and small population Wyoming communities will be an important factor in recruiting and keeping physicians in those communities. Since a major mission of the college is to provide practicing physicians for the State of Wyoming, I feel then that the provision of relevant continuing medical education in the community where the physician practices will be important in solving the problem of physician distribution. Further, the faculty of the College of Human Medicine will include specialists in educational technology and biomedical communications who could contribute significantly to a state-wide program coordinated with the Wyoming State Medical Society." Dr. Daugherty also stated that he intended to use the Continuing Education Committee of the Wyoming State Medical Society as the vehicle for the delivery of the Medical School's continuing education program to physicians throughout the state.

Closing the meeting, the committee chairman noted that "a joint coordinated effort involving all of the physicians of the state is one that will be most likely to serve the needs of the practicing physicians in Wyoming."



COLORADO

Fellows Named

Eight Colorado physicians were among the 373 new Fellows named to the American College of Physicians recently. They include R. William Hilty, MD, and Melvin R. Stjernolm, MD, both of Boulder; Ernest E. Miller, MD, and Bruce A. Pattee, MD, of Colorado Springs; Thomas M. Golbert, MD, of Denver; and D. Eugene Cowen, MD, Englewood.

Boulder Doctor Serves in Brazil

G. Robert Boyer, MD, Boulder, Colorado, has joined the Project HOPE medical education program in Maceio, Brazil, where he is serving as a pathology educator. This marks Dr. Boyer's return to Maceio where he had previously served with Project HOPE in 1973.

Denver Surgeons Present Papers

Papers and panels in which five Denver surgeons took part were presented during the recent 13th annual scientific meeting of the American Academy of Facial Plastic and Reconstructive Surgery, Inc. in Boston. Raymond P. Wood, II, MD, and Bruce B. Baker, MD, were co-authors of a paper entitled "A Single-Stage Dynamic and Static Fascia Lata Sling Procedure for Facial Paralysis" which was presented by the two authors.

A Panel discussion on "Anatomy of the Nose: External Support" was given by M. Dion, MD, Bruce W. Jafek, MD, and C. Tobin, MD. The sessions were attended by 1200 otolaryngolgists.

AAFP Names Diplomates

Joseph G. Sever, MD, and Clarence O. Hughes, MD, both of Englewood, have been named Diplomates of the American Board of Family Practice, as have Philip Hayes Norton, MD, Richard A. Patt, MD, and Ronald J. Swarsen, MD, all of Aurora. They are thereby certified in the specialty of family practice.

Fig. 1. Patty Sanborn of Fort Collins was first place over-all winner in the 22nd annual Colorado State Science Fair.

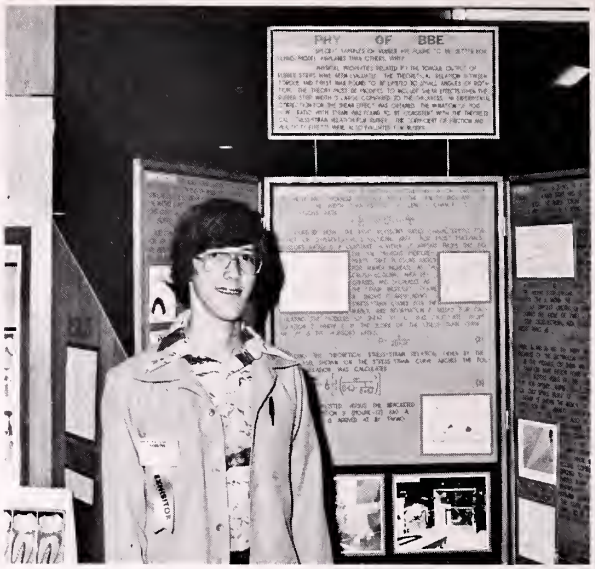


Fig. 2. Peter Sanborn of Fort Collins received second-place over-all winner for his entry on "Physics of Rubber."

Explorations of profound scientific matters continue to be made by young scientists in junior and senior high schools, largely inspired to original analyses and fresh observations by the annual Colorado State Science Fair, and the opportunity to proceed from it to the International Science Fair.

On April 14-16, 1977, 169 top students in Colorado participated in the 22nd Annual Colorado state science fair, held on the Blue Mall Balcony in the Cinderella City Shopping Center, Englewood, Colorado, during which 134 prizes were awarded.

Overall winner with a total of nine awards was 15-year old Patty M. Sandborn, 10th grader at Fort Collins High School at Fort Collins, whose exhibit entered in the Physical Science category described the "Mechanics of Insect Flight," an exhibit which was ajudged first place winner in the Senior High division.

Other commendations were those of the American Institute of Aeronautics and Astronautics, Rocky Mountain Section, which awarded Patty a check for $100; from The Denver Post she received a $250 book award and plaque; from the Massachusetts Institute of Technology Alumni of Colorado she received a subscription to *Technology Review;* the National Aeronautics and Space Administration awarded her a fourth place for her study; she received a first place award from the Society of Women Engineers, and a second place from the U.S. Navy. She also received an all-expense paid trip to the International Science Fair at Cleveland, Ohio in May.

Peter A. Sandborn, 11th grade student at Fort Collins High School, older brother of Patty, was second ranking winner in the overall category, with seven commendations including first place in the senior high engineering category. In addition he received $25 from the American Society of Mechanical Engineers, Colorado-Wyoming section; an award from the Hewlett-Packard Company, a subscription to *Junior Engineering,* from the Massachusetts Institute of Technology Alumni of Colorado an award for the outstanding Engineering presentation; a subscription to *Technology Review,* a certificate from the U.S. Army, and, received an all-expense paid trip to the International Science Fair in May in Cleveland, Ohio.

As in the previous 21 fairs, the Colorado State Science Fair was sponsored by the Colorado Medical Society, American Cancer Society, Colorado Division; Colorado Engineering Council, Colorado Medical Society Auxiliary, The Denver Post, National Bureau of Standards, National Oceanic and Atmospheric Administration, and Office of Telecommunications/Institute for Telecommunication Science, with the support of the Cinderella City Merchants Association.

Dr. Sam Shushan served as Fair Director, with Ralph F. Desch as Fair Chairman. Betty Whitmore is Executive Secretary, and Mrs. Eileen Irwin, Charles Bragow, and Dr. Calvin Fisher served as consultants.

In the Senior High Division, John Wiest, 12th grade student at Holyoke High School, Holyoke, placed first in the Botany judging with a report on the "Transmission of Infrared Light by Chlorophyll:" Wiest also received a year's subscription to Arctic and Alpine Research Journal from the Institute of Arctic and Alpine Research and a one year's subscription to *Technology Review* from the Massachusetts Institute of Technology Alumni of Colorado.

Justin Marble, 11th grade student at Arickaree School, Anton, placed first in Senior High mathematics for his exhibit on a "Universal Turing Machine," in which he developed a "hypothetical computer with unlimited data storage." He was awarded a years's subscription to the *Technology Review* by the Massachusetts Institute of Technology Alumni Association and a $75 bond for his first place.

Carl Hess, 11th grader at Boulder High School, Boulder, placed first in Senior High Zoology judging with his "Computer Ecology."

In the field of Botany in Junior High judging, Dianna Doherty, 9th grade student in Branson School, Branson, won a first place for a report on "Amino Acid identification in Prickly Pear Cactus-Opuntia chadantha."

There were several recommendations of students for college scholarships. Donald Bear of Hotchkiss was recommended for one to Western State College at Gunnison. Mary Lou Bond of Durango received a recommendation for Fort Lewis State College. Mark L. Burgus of Broomfield was recommended to Colorado School of Mines. Joe Haselhorst of Arvada was recommended as an alternate for a scholarship to the University of Colorado, as was Donna Haynie also of Arvada. Cindy Lykkehoy of Arickaree was given a recommendation for a scholarship to Northeast Junior College at Sterling. Ronald A. Sinton of Denver was recommended for a scholarship to the University of Colorado.

University of Colorado Medical Center

Silver and Gold Awards to Doctors

Lula Lubchenko, MD, Class of 1939, and Dale Atkins, MD, class of 1945, received the highest honor of the University of Colorado Medical Alumni Association in May. This honors doctors for outstanding community service, and for contributions to the science and art of medicine.

Dr. Lubchenko, co-director of the Newborn Service, pioneered in neonatology. She was born in Russia, and received her MD at the University of Colorado School of Medicine in 1939, and joined its faculty in 1943. In 1973 she was named "Medical Woman of the Year " by the National Board of the Medical College of Pennsylvania. She and her husband, Carl J. Josephson, MD, will retire July 1, but she will continue on a part-time basis to research several significant projects.

Atkins received his MD at the University of Colorado School of Medicine in 1945, and served for 12 years as Regent of the University of Colorado.

Zwillich Presents Paper

A paper on "Comparison of protein and carbohydrate meals on metabolism, ventilation and chemosensitivity" was presented by Clifford W. Zwillich, MD, assistant professor of medicine at the national meeting of the American Federation for Clinical Research in Washington, D.C.

Head and Neck Cancer Study Granted

A grant to train health professionals in prevention, diagnosis, and treatment of head and neck cancer for a three-year period will be financed at the University of Colorado Medical Center by a $117,000 grant from the National Cancer Institute.

Robert Greer, MD, received one of six grants from the NCI. He is assistant professor of pathology in the School of Medicine, and chairman of the Division of Oral Pathology and Oncology, and director of the Sands House Clinic in the School of Dentistry.

The primary objective is the training of physicians and allied health professionals, dental students, dental faculty, and practicing dentists in recognition, diagnosis, and treatment of discovered cancers.

Tan to Head Rheumatology

Eng M. Tan, MD, head of the Division of Allergy and Immunology of the Scripps Clinic and Research Foundation in La Jolla, California, has been named to head the Division of Rheumatology in the Department of Medicine at the University of Colorado Medical Center.

Dr. Tan received his MD from Johns Hopkins University School of Medicine, Baltimore, Maryland. He was a research fellow at Western Reserve for two years, and served three years at Rockefeller Institute, New York City. He fills the vacancy created when Charley Smyth, MD, became director of the Joe and Betty Alpert Arthritis Treatment Center at General Rose Memorial Hospital.

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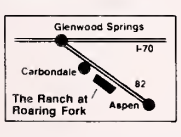

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University of New Mexico Medical School

New Mexico Dean Retires

Frank Land, MD, has resigned as associate dean for clinical affairs at the New Mexico School of Medicine to make a return to full-time family medicine instruction at the new family medicine department at Georgetown University, Washington, D.C.

Weston Named to Consult

James Weston, MD, medical investigator and pathology professor, is serving on an advisory committee to the City of New York, assisting in a review of operations of the Office of the Chief Medical Examiner in the City of New York.

Hendryson Scholarship Established

In honor of Irvin E. Hendryson, MD, a member of the faculty since 1967 who died in April, a scholarship to benefit an Indian student has been established at the School of Medicine. Dr. Hendryson had been a consultant to the Navajo Health Authority in Window Rock for the past several years. Memorials may be sent to the Office of the Dean, University of New Mexico School of Medicine, Albuquerque, New Mexico.

THE BOOK CORNER

New books received are acknowledged in this section and such acknowledgment must be regarded as sufficient return for the courtesy of the sender. Selection will be made for review in the interests of our readers and as space permits. Books are listed with advance data supplied by publishers. Prices quoted are not guaranteed. For further information, address queries to the publishers. Books here listed are available for lending from the Denver Medical Society Library.

Recent Acquisitions

Brief Scan of the Minutes of the Pueblo County Medical Society 1881-1976: Harvey Phelps. Pueblo, Colorado, 1976. 60 p. Gift.
Colorado Hospital Emergency Facilities Survey: Colorado/Wyoming Regional Medical Program. Denver, R.M.P., 1976. 206 p. Gift.
Drug Utilization Review in Skilled Nursing Facilities: U.S. Dept. H.E.W. Washington, D.C., G.P.O., 1975. Gift.
Hemoglobinopathies: Howard F. Bunn. Philadelphia, Saunders, 1977. 308 p. (Major problems in internal medicine, v. 12) $16.00.
The High Risk Infant: Lula O. Lubchenco. Philadelphia, Saunders, 1976. 294 p. (Major problems in clinical pediatrics, v. 14) $18.50.
Instructional Course Lectures: American Academy of Orthopaedic Surgeons. v. 25. St. Louis, Mosby, 1976. 247 p. $27.50.
Insurance Handbook for the Medical Office: Marilyn T. Fordney. Philadelphia, Saunders, 1977. 646 p. $12.95.
Malabsorption: Marvin H. Sleisenger. Philadelphia, Saunders, 1977. 261 p. (Major problems in internal medicine, v. 13).
Pathophysiology: Edward D. Frohlich, ed. 2nd ed. Philadelphia, Lippincott, 1976. 902 p.
The Physician's Office: Leif C. Beck. Princeton, N.J., Excerpta Medica, 1977. 170 p. $14.95.
Proceedings of the International Conference on Women in Health: Health Resources Administration. Washington, D.C., H.R.A., 1976. 204 p. Gift.
Planning Guide for Physician Medical Facilities: American Medical Association. 2nd ed. Chicago, A.M.A., 1975. 34 p. $2.00.
The Sclera and Systemic Disorders: Peter G. Watson. Philadelphia, Saunders, 1976. 458 p. (Major problems in ophthalmology, v. 2) $20.00.
Solid Liver Tumors: James H. Foster and Martin M. Berman. Philadelphia, Saunders, 1977. 342 p. (Major problems in clinical surgery, v. 22) $18.00.
Southwestern Medical Dictionary: Margarita A. Kay, et. al. Tucson, Arizona, Univ. of Arizona Press, 1977. 217 p. Review book.
Surgery in the United States: American College of Surgeons and American Surgical Association. Chicago, A.C.S. & A.S.A., 1975. 207 p. Gift.
Statistical Abstract of Colorado: Thomas G. Tyler,. Denver, Transrep Bibliographics, 1977. 515 p. $15.00.1

Book Review

Psychosocial Counseling in General Medical Practice: Allen Hodges, Lexington Books, Lexington, Massachusetts, 1977, 116 p.

This book, written by a man with an impressive experience and background in both public health and mental health, deals basically with the art of healing, or more precisely, the loss of that part of the art, as many of us who are involved in the clinical practice of medicine see it. The author stresses the need for the primary physician to re-introduce in his total care plan a basic understanding of his patients' psychologic and social problems, and to systematically incorporate these findings in his total treatment program. In an age of increased specialization, medical care has often become depersonalized, leading to frustration both on the part of the patient as well as the practitioner. Dr. Hodges, who in this book attempts to find an answer to reintegrate the physical and biological with the emotional aspects of health, illustrates this point with many interesting case histories based on a fruitful working relationship with a clinical physician, Dr. Stewart L. Smith. In order to bring together the medically oriented thinking of the physician with the psychosocial knowledge of the psychologist the author draws heavily on two concepts recently developed in the two disciplines. Starting out with Weed's Problem-Oriented-System, he makes a good case for integrating into this system a workable psychosocial problem finding approach by the use of the Minnesota Multiphasic Personality Inventory Test.

The book is well illustrated with a large number of case histories which deal with such common problems of the primary physician as depression and grief, marital problems, psychoneurosis and general problems in life style. The author sees the MMPI test as an ideal answer because of its non-psychiatric jargon to assist the family physician in getting a quick insight into some of the problem areas confronting his patients as the effect of his psychosocial environment. The book ends with a very readable and interesting short treatise on the domain of health and makes a case for future areas of cooperation between the physician and the psychologist.

My only criticism would be that Dr. Hodges in his 28 years of careers in various levels of public health and mental health has developed a natural grasp for a working team approach which is not necessarily true for the average clinical physician. Primary physicians who have not had a close exposure to the clinical psychologist may find it difficult to develop a meaningful communication and instead may take it upon themselves to start using the MMPI test on their own without sufficient knowledge of the possible pitfalls this test can cause.

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	29-Sept. 2	Seattle, Washington	706
September	7-11	Ann Arbor, Michigan	707
	7-11	Richmond, Virginia	708
	~~7-11~~	~~San Francisco, California~~ **sold out**	~~709~~
	14-18	Kansas City, Missouri	710
	~~26-30~~	~~Cambridge, Massachusetts~~ **sold out**	~~711~~
	26-30	Los Angeles, California	712
	26-30	Philadelphia, Pennsylvania	713
	26-30	Rochester, Minnesota	714
	26-30	Washington, DC	715
October	1-5	Montauk Point, L.I., NY	716
	3-7	Cincinnati, Ohio	717
	3-7	Denver, Colorado	718
	3-7	Houston, Texas	719
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Colorado Medical Society
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$35 per person registration fee ($20 for full-time students and Senior Citizens) which includes two continental breakfasts and two luncheons. Make checks payable to Colorado Medical Society. Room reservations should be made directly with Lion Square, 655 W. Lionshead Place, Vail, Colorado, 81657.

MEETINGS

University of Colorado School of Medicine
Continuing Medical Education

PRACTICAL NEUROLOGY FOR THE INTERNIST AND FAMILY PHYSICIAN
The Given Institute of Pathobiology

July 11-13, 1977

* * *

GASTROENTEROLOGY: CURRENT CLINICAL TOPICS
The Gant, Aspen, Colorado

July 11-15, 1977
Credit: Approved for AMA Category 1 credit.

* * *

HEMATOLOGY-ONCOLOGY
The Given Institute of Pathobiology, Aspen, Colorado

July 14-17, 1977
Credit: Approved for AMA Category 1

* * *

GYNECOLOGY
The Mark, Vail, Colorado

July 17-20, 1977
Credit: Approved for AMA Category 1

* * *

INTERNAL MEDICINE
YMCA of the Rockies, Estes Park, Colorado

July 18-22, 1977
Credit: Approved for AMA Category 1

PEDIATRICS
The Given Institute of Pathobiology, Aspen, Colo.

July 24-27, 1977
Credit: Approved for AMA Category 1

* * *

PATHOLOGY IN PEDIATRICS
The Gant, Aspen, Colorado

July 25-29, 1977
Credit: Approved for AMA Category 1

* * *

PEDIATRIC DERMATOLOGY
The Given Institute of Pathobiology, Aspen, Colorado

July 27-29, 1977

Approved: For AMA Category 1

* * *

PATHOLOGY IN GYNECOLOGY AND OBSTETRICS
The Given Institute of Pathobiology, Aspen, Colorado

August 6-10, 1977
Credit: Approved for AMA Category 1

* * *

CLINICAL OTOLARYNGOLOGY FOR THE PRIMARY CARE PHYSICIAN
The Mark at Vail, Colorado

August 7-10, 1977
Credit: 26 hours Category II for Physician's Recognition Award-AMA

* * *

DISEASES OF THE KIDNEY AND RENAL FAILURE
The Given Institute of Pathobiology

August 10-13, 1977
Credit: Approved for AMA Category 1
Contact: Office of Postgraduate Medical Education, University of Colorado Medical Center, Container C295, 4200 East 9th Avenue, Denver, Colorado 80262

* * *

WIDENING SCOPE OF DYNAMIC PSYCHIATRY
The Given Institute of Pathobiology, Aspen, Colorado

August 22-26, 1977
Credit: Approved for AMA Category I

* * *

14th ANNUAL HOSPITAL MEDICAL STAFF CONFERENCE
THE MARK, Vail, Colorado

September 7-10, 1977
Credit: 18 hours AMA Category I.

* * *

2ND ANNUAL SYMPOSIUM ON VASCULAR SURGERY
The Marriott Hotel, Denver, Colorado

September 29-October 1, 1977
Credit: Approved for 20 hours of AMA Category I
Contact: Office of Postgraduate Medical Education, University of Colorado Medical Center, Container C295, 4200 East 9th Avenue, Denver, Colorado 80262.

Symposia de Santa Fe
SELECTED TOPICS IN HEMATOLOGY AND ONCOLOCY

La Fonda Hotel, Santa Fe, New Mexico

July 14-16, 1977

* * *

HYPERALIMENTATION AND FLUID THERAPY FOR SURGEONS

Inn at Loretto, Santa Fe, New Mexico

August 11-13, 1977

* * *

RADIOLOGY FOR FAMILY PRACTITIONERS

Hilton Inn, Santa Fe, New Mexico

September 14-16, 1977

Contact: Walter J. Levy, MD, P.O. Box 5175, Coronado Station, Santa Fe, New Mexico 87502.

The American Academy of Family Physicians, New Mexico Chapter
20TH ANNUAL RUIDOSO FAMILY PRACTICE SEMINAR

Inn of the Mountain Gods, Mescalero, New Mexico

July 18-21, 1977
Credit: 17 hours AAFP Prescribed, and 17 hours Category 4, New Mexico Board of Medical Examiners.
Contact: Bob Reid, P.O. Box 456, Sunland Park, New Mexico 88063.

Montana Children's Home and Hospital
ETHICS-MORALS SEMINAR on GENETIC AND BIRTH DEFECTS

Carroll College, Helena, Montana

July 25-26, 1977
Contact: Philip D. Pallister, MD, P. O. Box 1715, Helena, Montana 59601

New Mexico Heart Institute - Presbyterian Hospital Center
6TH ANNUAL SANTA FE CARDIOLOGY SYMPOSIUM

The Inn at Loretto, Santa Fe, New Mexico

August 5-6, 1977
Credit: Approved for 12 (Prescribed Elective) hours by AAFP.
Contact: Santa Fe Cardiology Symposium, c/o Presbyterian Hospital Center, Albuquerque, New Mexico 87102.

Beth Israel Hospital
ASPEN MUSHROOM CONFERENCE

Hotel Jerome, Aspen, Colorado

August 7-12, 1977
Credit: AMA Physician Recognition Award, Up to 30 hours Category II.
Contact: Aspen Mushroom Conference, **c/o Beth Israel Hospital, 1601 Lowell Blvd., Denver, Colorado 80204.**

The Menninger Foundation
PHYSICIANS AND THEIR FAMILIES

August 14-19, 1977
YMCA Camp, Estes Park, Colorado

Credit: Approved for 25 hours, Category I, Physician's Recognition Award-AMA
Contact: Erwin T. Janssen, MD, The Menninger Foundation, Box 829, Topeka, Kansas 66601

American Association of Diabetes Educators
4TH ANNUAL MEETING/EDUCATIONAL WORKSHOP

Hilton Hotel, Denver, Colorado

August 14-16, 1977
Contact: Kate Doyle, AADE, 3553 W. Peterson Ave., Chicago, Illinois 60659.

American Society for Surgery of the Hand
INTERNATIONAL HAND SURGERY SYMPOSIUM

August 15-19, 1977
THE MARK, Vail, Colorado
Credit: Approved for 18 hours Category I, Physician's Recognition Award-AMA
Contact: John A. Boswick, Jr., MD, 4200 East 9th Avenue, C309, Denver, Colorado 80262

American College of Cardiology
TUTORIALS IN THE TETONS: CARDIAC EMERGENCIES

August 21-23, 1977
Jackson Lake Lodge, Grand Teton National Park, Moran, Wyoming
Credit: Approved for 12 hours Category I, Physician's Recognition Award-AMA.
Contact: Miss Mary Anne McInerny, American College of Cardiology, 9650 Rockville Pike, Bethesda, Maryland 20014

American Cancer Society
2ND NATIONAL CONFERENCE ON HUMAN VALUES & CANCER

September 7-9, 1977
The Palmer House, Chicago, Illinois

The Children's Hospital
THE 3RD ANNUAL BURN SYMPOSIUM

Tammen Auditorium, The Children's Hospital, 1056 East 19th Avenue, Denver, Colorado

September 9, 1977
Contact: Wm. Carl Bailey, MD, Director, Burn Team, The Children's Hospital, Denver, Colorado, 80218, or call: (303) 825-8289

NATIONAL CONFERENCE ON THE LYMPHOMAS AND THE LEUKEMIAS
Waldorf-Astoria Hotel, New York City, New York

September 29-October 1, 1977
Credit: 15-½ Elective Hours AAFP
Contact: American Cancer Society, 777 Third Avenue, New York City, New York 10017

American Medical Association
37TH ANNUAL AMA CONGRESS ON OCCUPATIONAL HEALTH

Stouffer's Riverfront Towers, St. Louis, Missouri

September 19-20, 1977
Credit: Approved for 12 hours Category I, Physician's Recognition Award-AMA.
Contact: Department of Environmental, Public, and Occupational Health, AMA, 535 North Dearborn Street, Chicago, Illinois 60610.

Colorado Medical Society
107TH ANNUAL MEETING

September 21-25, 1977
The Broadmoor, Colorado Springs, Colorado

Montana Medical Association
99TH ANNUAL MEETING

September 29-October 1, 1977
Village Motor Inn, Missoula, Montana

* * *

American Association of Electromyography and Electrodiagnosis
24TH ANNUAL MEETING

Hilton Hotel, Salt Lake City, Utah

September 30-October 1, 1977

Contact: Michael Cherington, MD, University of Colorado Medical Center, Department of Neurology, 4200 East 9th Avenue, Denver, Colorado 80220

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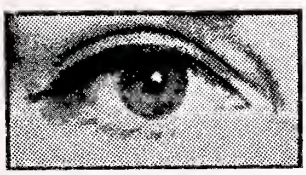

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Publication rules and suggestions to authors

1. Who may submit articles. The Rocky Mountain Medical Journal ordinarily accepts only articles prepared by members of the state societies we serve and by guest speakers at their official meetings.

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Title (preferably short) and author's name, city and state must appear at the top of the first page. Second and subsequent pages should be identified with consecutive page numbers and author's surname. If physician authors possess more than one degree, the MD only is used. Lesser degrees of non-MD authors or co-authors may be used.

Please include beneath the title or at the top of the first page a brief paragraph stating what the article is designed to show and, therefore, why it should be read. This introductory feature is, in editorial parlance, called the "blurb"; it is printed in bold type.

3. Where to submit material. All copy must be sent to the editor for the state in which the material originates. Editors for each state, with their mailing addresses, are listed monthly on the Table of Contents page of the Journal.

4. Acceptance or rejection. The state editor will (1) tentatively accept the article, (2) return it to the author with suggestions for revision, or (3) reject it. He will edit and forward approved copy to the publication office in Denver for final editing and scheduling.

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6. Length of articles. Scientific papers should be "boiled down" not to exceed ten pages of double-spaced typewritten standard 8½ x 11 inch sheets. Shorter papers are more acceptable to editors and readers, and earlier publication is probable. Every journal is in competition for the reader's time, and condensation is the order of the day. For this reason, and because of financial and space limitations, we request that copy be condensed to the greatest extent consistent with conveying the message.

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12. Editorials. We would like to have more submitted by our State editors and the membership at large. Readers must get tired of thunder from publication headquarters in Denver; frankly, we do too! Speak out, and we'll sign your name, initials, or respect your request for anonymity, as you choose. If you don't like something we do or say, please let us in on it; we could even stand to hear about it if something happens to please you. A "Letter to the Editor" may help you get something off your mind. We're asking for it and we have a place to put it—in the Journal, that is!

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OBITUARIES

Colorado

Doctor Irvin E. Hendryson, known as "Chief," to his intimates, died after a long illness at his home in Albuquerque, New Mexico, Monday, April 11, 1977. An orthopedic surgeon of renown, having contributed substantially to professional journals relating to his field of interest, he was perhaps best known for his untiring dedication to organized medicine.

He served on numerous committees of the Denver and Colorado medical societies, and in 1957 was elected president of the Denver Medical Society. He was an Alternate Delegate to the American Medical Association from the Colorado Medical Society from 1954 to 1958, becoming a Delegate in 1958, in which capacity he served until 1965. He also served on numerous committees, including the Committee on Medical Facilities, the Council on Legislative Activities, the Council on Medical Service, the National Speakers Bureau, and the Emergency Medical Service Committee.

He was on the Board of Trustees of the American Medical Association from 1965 to 1970. During most of this time he maintained an active interest in his local and state medical societies, as well as community and health organizations.

A graduate of the University of Colorado, he served on the faculty of that institution as Assistant Clinical Professor of Surgery for 20 years. He moved to Albuquerque to join the faculty of the University of New Mexico as Associate Professor of Surgery and Director of Emergency Services. He also served as Assistant Dean of that institution for a brief period. He became involved in providing medical services to the Navajo reservation at Window Rock, Arizona.

Among many positions he filled, he was consultant on medical devices to the Food and Drug Administration, and served as trustee for the National Council on Alcoholism. He was medical director of the National Ski Patrol System.

In addition to his membership in the local and state medical societies and the American Medical Association, he belonged to the American Academy of Orthopedic Surgeons and the American Academy of General Practice (sustaining member). He was certified by the American Board of Orthopedic Surgeons in 1947.

Doctor Hendryson is survived by his widow Elizabeth, by three daughters, Mrs. Susan Nye of Chattanooga, Tennessee; Miss Jane Hendryson of Palo Alto, California, and Mary Ann Hendryson, Denver, and two sons, R. Michael of Palo Alto, California, and John of Breckenridge, Colorado. Mrs. Elizabeth Weld, Beverly Massachusetts, a stepdaughter, and Dr. Peter New, Gloucester, Massachusetts, a step-son, also survive.

Doctor T. Donald Cunningham died in Denver April 24, 1977 after a long illness, at the age of 87. In 1921 he became associated in practice with the then renowned J. N. Hall, MD, starting his long and eventful medical career.

Earlier he had achieved recognition as a leader of the East Denver high school football team, sparking it to championships in the Rocky Mountain Football Conference for three successive years.

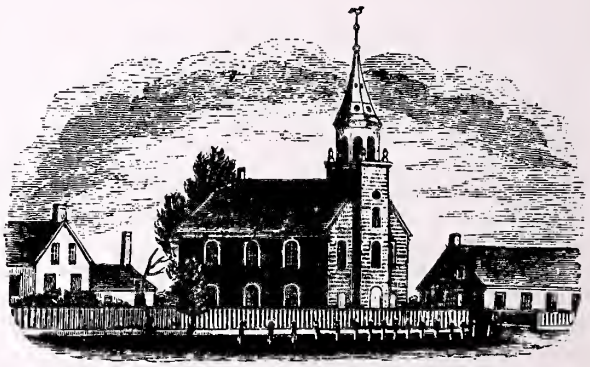
After receiving his BS degree from Dartmouth in 1913, he entered the Harvard University Medical School. In 1917, as a senior, he joined the Harvard Surgical Unit attached to the British army and served overseas for several months. After returning to Harvard for studies he received his MD in 1918, following which he joined the U.S. Medical Reserve Corps.

After an internship at Massachusetts General Hospital he served a residency at Peter Bent Brigham Hospital in Boston. Before entering solo practice he spent three years with Dr. Hall in Denver. He became a consultant with a special interest in allergic disease. In 1928 he joined the faculty of the University of Colorado School of Medicine, serving until 1934 when he was an Assistant Clinical Professor.

He was certified in 1937 by the American Board of Internal Medicine, in both Medicine and Allergy. In addition to membership in the Denver and Colorado medical societies and the American Medical Association, he held memberships in the Central Society for Clinical Research, the Society for the study of Asthma, and Allied Conditions, Phi Rho Sigma and Sigma Xi fraternities. He was a Fellow of the American College of Physicians.

He belonged to the Denver Club, Denver Country Club, and the Masonic Lodge.

Doctor Cunningham is survived by his widow Barbara, two sons, Thomas Donald, Jr., of Bedford, New York and Dr. Charles C. of Springfield, Vermont, and a sister, Mrs. Ivan D. Wood of Denver.



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Milk, like many foods today, is being looked at much more closely than ever before by physicians and nutritionists.

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A child between the ages of six months and six years, for example, can get at least three-fourths of his daily dietary allowance for calcium, riboflavin, vitamins D and B_{12}, phosphorous, and protein from just three glasses of milk. And milk is also a good source of vitamins A and B_6, as well as thiamin and niacin.

That's why the U.S.D.A., acting on the recommendation of the National Research Council—National Academy of Sciences, has established these guidelines for milk consumption:

Children up to 6 years
Two to three 8 oz. glasses per day.

Children 6 to 12 years
Three or more 8 oz. glasses per day.

For a free copy of the Dairy Council brochure "Composition and Nutritive Value of Dairy Foods," contact Dairy Council, Inc., 12450 N. Washington, Thornton, CO 80241.

Per cent of Recommended Daily Allowance contributed by three 8 oz. glasses of fortified milk.

	6 mos.—1 yr.	1 yr.—3 yrs.	3 yrs.—6 yrs.
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Riboflavin	100	100	100
Vitamin B_{12}	100	100	100
Protein	100	100	80
Phosphorus	100	85	85
Vitamin B_6	76	51	34
Vitamin D	75	75	75
Niacin*	71	63	47
Thiamin	56	40	31
Vitamin A	46	46	37

*maximum niacin equivalents based on: 60 mg tryptophan=1 mg niacin

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The population of McCook is 10,000, with approximately 25,000 people in the broad service area. Cattle feeding, grain production, and industry create a well balanced economy.

CONTACT: **KEN NOTEBOOM, Administrator**
McCook Community Hospital
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WANT ADS

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FAMILY PRACTITIONER WANTED TO SHARE OFFICE. Well-established office in SE Denver for 13 years. Moving by January 1978 to larger space. Write Box 577-2-TFB co Rocky Mountain Medical Journal, 1601 East 19th Avenue, Denver, Colorado 80218. 577-2-TFB

PHYSICIAN ASSISTANT seeks position with pediatrician. 1977 graduate of Western Michigan University with BS. AMA-approved. BS in child development and teaching. Four years as certified respiratory therapy technician, including special training for neonates. Write Kathleen Nitz, 709 W. Lovell, #2, Kalamazoo, Michigan, 49007. 377-13-3B

PRIMARY CARE PHYSICIAN'S ASSISTANT. Graduates from Western Michigan University August 1977 with BSM, AMA approved. Relocating in Colorado, seeks small group practice that could utilize a PA. BS - University of Wisconsin, four years experience Corpsman in USN. Write Donald Faulkner, 1605 W. North Street, Kalamazoo, Michigan, 49007. 377-14-3B

CIVILIAN PHYSICIANS needed in all specialties at Army Medical Centers and major hospitals and clinics throughout USA. Overseas tours available at later date. Full Civil Service benefits including liberal leave. 40 hour work week, malpractice protection and retirement eligibility, after five years. Credit given for prior military service. Salary: $30 to 38K. IN DENVER: GP (Emergency Room), Occupational Health and Pediatrics skills needed. IN COLORADO SPRINGS: Radiologist. Contact: R.P. Johnson, Box 484 LAMC Presidio of San Francisco, California 94129 or call: (415) 561-3531. Collect. Non-Discrimination Employment Practices. 577-11-3B

WANTED: Surgeon with FP or GP interest to join a growing rural group practice in Northwest Montana. Modern medical clinic and 30-bed hospital. Mountainous setting with ideal climate. Foundation sponsorship. No investment required. Fringe benefits. Write: P.A. Hinton, Administrator, or J. Michael Wise, MD, Medical Director, Mission Valley Health Services, St. Ignatius, Montana 59865. 577-6-2B

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COLORADO: Board Certified General Surgeon for community of 7,000 in high plains of eastern Colorado. Will need to do some general practice. Guaranteed minimum $40,000 first year. Contact: Sarah Y. Bowman, Rural Health Care Association, 1565 Clarkson, Denver, Co. 80218 (303) 832-3135 777-7-1B

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ASSOCIATE WANTED: General surgeon willing to do some Family Practice. Suburban Denver area. Fully equipped office with laboratory and X-ray. Send resume to Box 777-9-1B, c/o Rocky Mountain Medical Journal, 1601 East 19th Avenue, Denver, Colorado 80218. 777-9-1B

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GASTROENTEROLOGIST AND NEUROLOGIST needed for multispecialty group of 48 physicians. A unique opportunity to join a vigorous group in an excellent location. Write or call: Paul V. Hoyer, MD, Medical Director, The Billings Clinic, P. O. Box 2555, Billings, Montana 59103. (406) 252-4141 777-14-2B

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PHYSICIANS' ASSISTANT seeks primary care position. May 1977 graduate of AMA-approved University of Iowa program with BSH. BA in biology from Lawrence University, with extensive research experience and some hospital care experience. Write: Elizabeth Coyte, 124 East Rusholme, Davenport, Iowa 52803, or call: (319) 324-4265. 777-18-1B

FOR SALE: Medical practice in internal medicine and rheumatology, including medical and office supplies, and equipment of recently deceased physician. Supplies and equipment in leased space, which will be available. Contact: Edwin S. Culver, Attorney at Law, 760 Sherman Street, Denver, Colorado 80203, or call: (303) 831-8307. 777-19-1B

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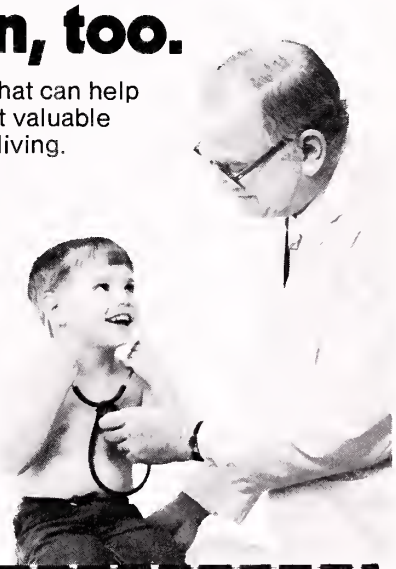

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Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma, may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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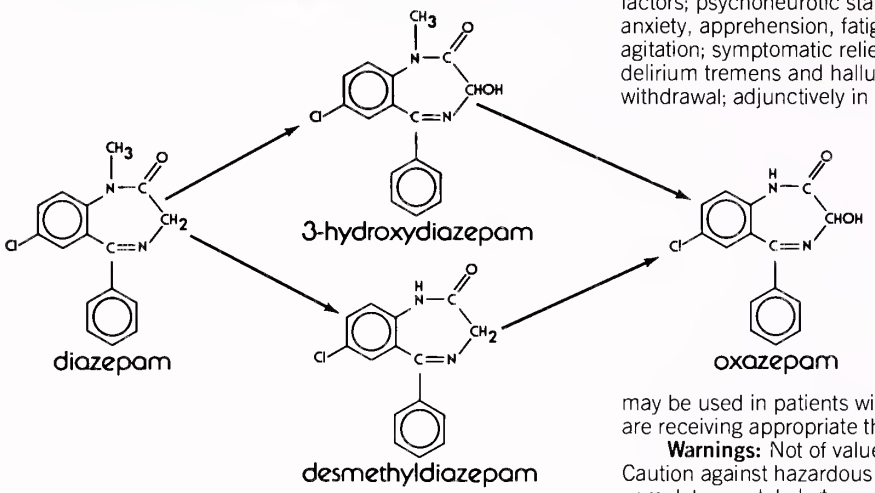
A pharmacokinetic character all its own



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Valium® (diazepam) IV

2-mg, 5-mg, 10-mg scored tablets

a prudent choice in psychic tension and anxiety

Before prescribing, please consult complete product information, a summary of which follows:

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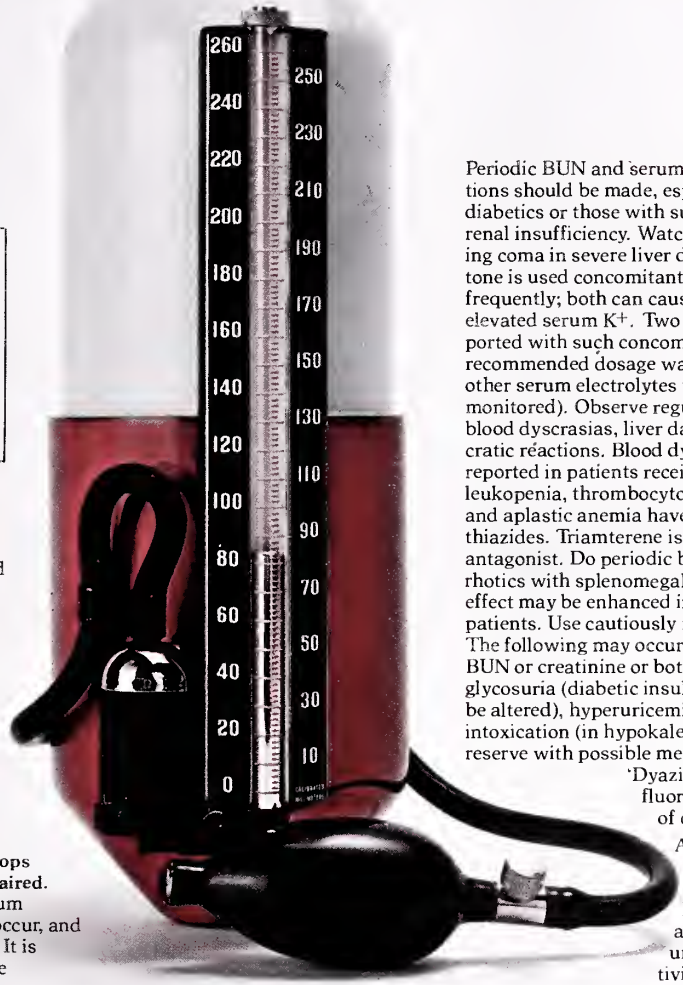

Before prescribing, see complete prescribing information in SK&F Co. literature or PDR. A brief summary follows:

* **Warning**
This drug is not indicated for initial therapy of edema or hypertension. Edema or hypertension requires therapy titrated to the individual. If this combination represents the dosage so determined, its use may be more convenient in patient management. Treatment of hypertension and edema is not static, but must be reevaluated as conditions in each patient warrant.

* **Indications:** When the combination represents the dosage determined by titration: Adjunctive therapy in edema associated with congestive heart failure, hepatic cirrhosis, the nephrotic syndrome. Corticosteroid and estrogen-induced edema, idiopathic edema; hypertension, when the potassium sparing action of triamterene is warranted. (See Box Warning.) Routine use of diuretics in healthy pregnant women is inappropriate; they are indicated in pregnancy only when edema is due to pathological causes.

Contraindications: Further use in anuria, progressive renal or hepatic dysfunction, hyperkalemia. Pre-existing elevated serum potassium. Hypersensitivity to either component or other sulfonamide-derived drugs.

Warnings: Do not use potassium supplements, dietary or otherwise, unless hypokalemia develops or dietary intake of potassium is markedly impaired. If supplementary potassium is needed, potassium tablets should not be used. Hyperkalemia can occur, and has been associated with cardiac irregularities. It is more likely in the severely ill, with urine volume less than one liter/day, the elderly and diabetics with suspected or confirmed renal insufficiency. Periodically, serum K^+ levels should be determined. If hyperkalemia develops, substitute a thiazide alone, restrict K^+ intake. **Associated widened QRS complex or arrhythmia requires prompt additional therapy.** Thiazides cross the placental barrier and appear in cord blood. Use in pregnancy requires weighing anticipated benefits against possible hazards, including fetal or neonatal jaundice, thrombocytopenia, other adverse reactions seen in adults. Thiazides appear and triamterene may appear in breast milk. If their use is essential, the patient should stop nursing. Adequate information on use in children is not available.

Precautions: Do periodic serum electrolyte determinations (particularly important in patients vomiting excessively or receiving parenteral fluids). Periodic BUN and serum creatinine determinations should be made, especially in the elderly, diabetics or those with suspected or confirmed renal insufficiency. Watch for signs of impending coma in severe liver disease. If spironolactone is used concomitantly, determine serum K^+ frequently; both can cause K^+ retention and elevated serum K^+. Two deaths have been reported with such concomitant therapy (in one, recommended dosage was exceeded, in the other serum electrolytes were not properly monitored). Observe regularly for possible blood dyscrasias, liver damage, other idiosyncratic reactions. Blood dyscrasias have been reported in patients receiving triamterene, and leukopenia, thrombocytopenia, agranulocytosis, and aplastic anemia have been reported with thiazides. Triamterene is a weak folic acid antagonist. Do periodic blood studies in cirrhotics with splenomegaly. Antihypertensive effect may be enhanced in post-sympathectomy patients. Use cautiously in surgical patients. The following may occur: transient elevated BUN or creatinine or both, hyperglycemia and glycosuria (diabetic insulin requirements may be altered), hyperuricemia and gout, digitalis intoxication (in hypokalemia), decreasing alkali reserve with possible metabolic acidosis. 'Dyazide' interferes with fluorescent measurement of quinidine.

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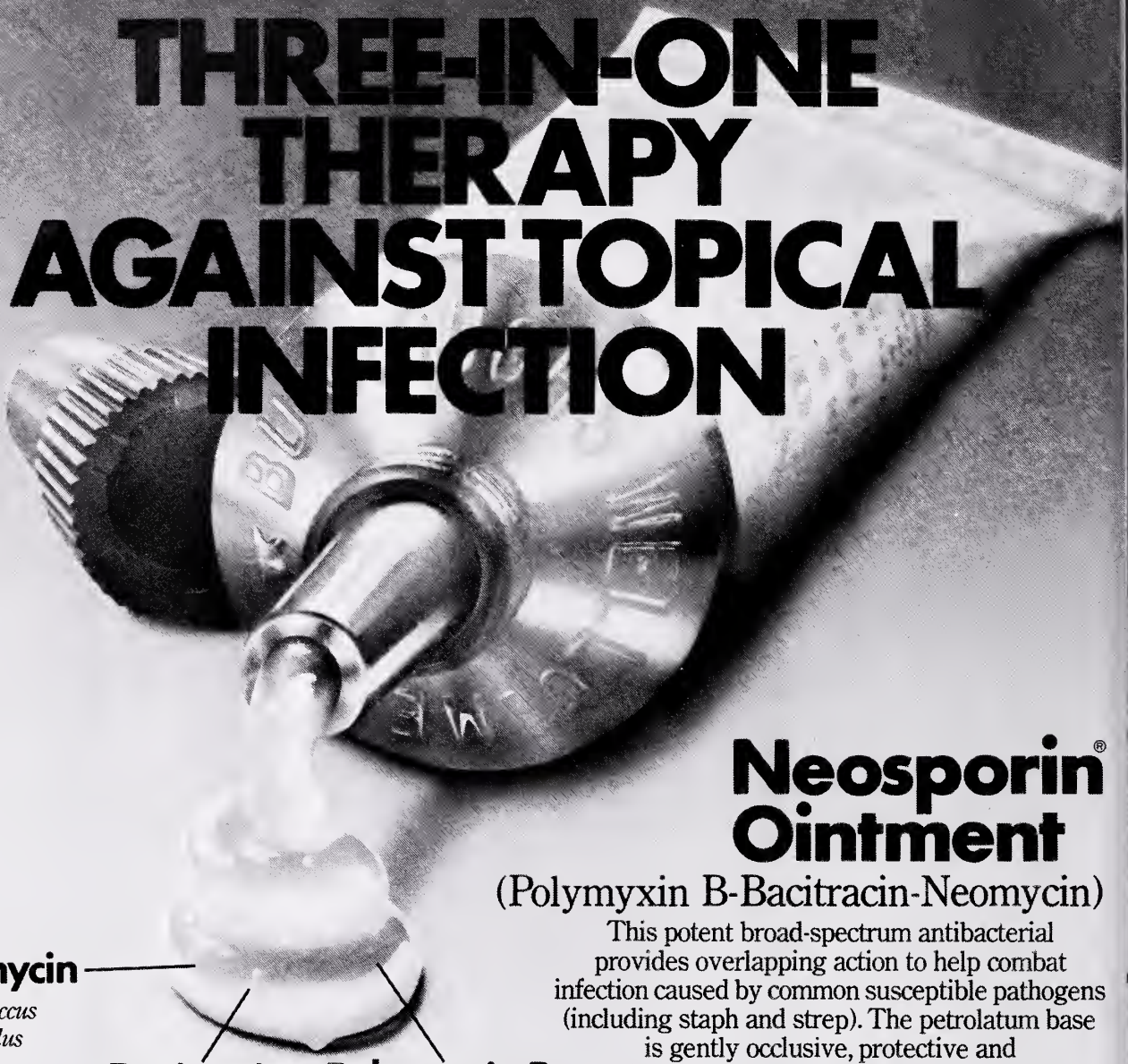

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Each gram contains: Aerosporin® brand Polymyxin B Sulfate 5,000 units; zinc bacitracin 400 units; neomycin sulfate 5 mg (equivalent to 3.5 mg neomycin base); special white petrolatum qs; in tubes of 1 oz and 1/2 oz and 1/32 oz (approx.) foil packets.

WARNING: Because of the potential hazard of nephrotoxicity and ototoxicity due to neomycin, care should be exercised when using this product in treating extensive burns, trophic ulceration and other extensive conditions where absorption of neomycin is possible. In burns where more than 20 percent of the body surface is affected, especially if the patient has impaired renal function or is receiving other aminoglycoside antibiotics concurrently, not more than one application a day is recommended.

When using neomycin-containing products to control secondary infection in the chronic dermatoses, it should be borne in mind that the skin is more liable to become sensitized to many substances, including neomycin. The manifestation of sensitization to neomycin is usually a low grade reddening with swelling, dry scaling and itching; it may be manifest simply as failure to heal. During long-term use of neomycin-containing products, periodic examination for such signs is advisable and the patient should be told to discontinue the product if they are observed. These symptoms regress quickly on withdrawing the medication. Neomycin-containing applications should be avoided for that patient thereafter.

PRECAUTIONS: As with other antibacterial preparations, prolonged use may result in overgrowth of nonsusceptible organisms, including fungi. Appropriate measures should be taken if this occurs.

ADVERSE REACTIONS: Neomycin is a not uncommon cutaneous sensitizer. Articles in the current literature indicate an increase in the prevalence of persons allergic to neomycin. Ototoxicity and nephrotoxicity have been reported (see Warning section).

Complete literature available on request from Professional Services Dept. PML.

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Rocky Mountain Medical Journal

COLORADO
MONTANA
NEW MEXICO
WYOMING

September-October 1977
VOLUME 74 • NUMBER 5

OUR COVER: Leaves aflame amidst the crispening fall air are what we choose to recall of autumn. In the high country near the Maroon Bells above Aspen the call of the outdoors sings out rapturously in Don Miller's splendid photo for which we are happy to give him credit.

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...LBY PROCLAIMS WOMAN SUFFRAGE

Signs Certificate of Ratification at His Home Without Women Witnesses.

MILITANTS VEXED AT PRIVACY.

Wanted Movies of Ceremony, But Both Factions Are

...SHINGTON, Aug. 26, 1920—...struggle for Wom...

Social Security Bill Is Signed; Gives Pensions to Aged, Jobl

Roosevelt Approves Message Intended to Benefit 30,0... Persons When States Adopt Cooperating Laws—He ... the Measure 'Cornerstone' of His Economic Progra...

WASHINGTON, Aug. 1... The Social Security Bill, po... a broad program of unemplo... insurance and old age p... and counted upon to bene... 20,000,000 persons, becam... day when it was signed b... dent Roosevelt in the pre... those chiefly responsible ... ting it through Congress.

Mr. Roosevelt called the ... "the cornerstone in a s... which is being built but ... means ...

SENATE APPROVES 18-YEAR OLD VOTE IN ALL ELECTIONS

Amendment to Constitution is Sent to House, Where Passage is Expected

WASHINGTON, March 10, 1971—The Senate approved today, 94 to 0, and sent to ...

...UL... ...IGNI...

TRUMAN CLOSES ...ITED NATIONS CONFEREN... ...VITH PLEA TO TRANSLATE CHARTER INTO DEEDS

NEW WORLD HOPE

President Hails 'Great Instrument of Peace,' Insists It Be Used

HISTORIC LANDMARK

Meeting Gives Standing Ovation as Executive Pictures Peace Gain

SAN FRANCISCO, June 26, 1945

"If we fail to use it," he declared to the solemn final meeting of the delegates, 'we shall betray all of those who have died in order that we might meet here in freedom and safety to create it.'

"If we seek to use it selfishly—for the advantage of any one nation or any small group of nations—we shall be equally guilty of that betrayal."

Fervent Interpolation

The President, speaking in the auditorium of the War Memorial Opera House, built in memory of sons of the Golden Gate city who gave their lives in the first World War, in which he himself served, seemed to give unconscious expression to the solemn feeling of the occasion when, at the outset of his speech, he interpolated the words, half a hope, half a prayer:

"Oh, what a great day this can be in history!"

Just before the plenary session the President accompanied the eight United States delegates to the auditorium of the Veterans'

...the Dra... ...Ends No...

WASHINGTON, Jan. 27, 1973—"With the signing of the peace agreement in Paris today, and after receiving a report from the Secretary of the Army that he foresees no need for further inductions, I wish

PATIENT PACKAGE INSERTS: A CONCEPT WHOSE TIME HAS COME?

The consumer's right to know is an irreversible and desirable trend of the Seventies. It extends, and properly, to a patient's right to know more about his or her prescription medications. One way, gaining favor, is through patient package inserts. Wisely-prepared and properly distributed when medically indicated, they could markedly improve patient knowledge and drug therapy—laudable goals by anyone's standards.

The PMA endorses these goals and will work with government, the health professions and consumers to achieve them.

The Advantages

The concept holds promise of benefits: better patient understanding of the product prescribed, better adherence to the treatment plan, and more awareness of possible side reactions.

Every doctor has had patients who fail to finish antibiotic regimens because they feel better. Some patients assume that if one tranquilizer or analgesic is good, two may be twice as good. Still others fail to report dizziness while on antihypertensive therapy —and so on.

Problems like these might arise less often if the patient received written information in addition to verbal instructions. Some studies suggest that patients are more receptive to such materials, and they more often understand the verbal instructions and follow them, when inserts are used.

The Disadvantages

There are also some potential problems. Obviously, the inserts must be clearly phrased, without extraneous or complex detail. How much information is enough? How can it be kept current? Should all patients receive the same information? Should inserts be included with all drugs? Should only potential problems be listed or are patients better off with a "fair balance" presentation that describes usefulness as well as drawbacks?

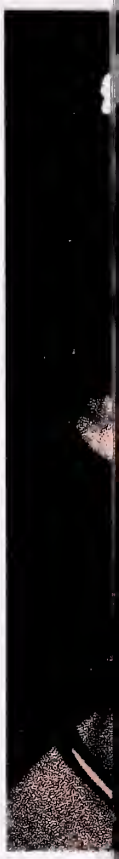
These and similar questions require answers, since model inserts have yet to be properly developed and tested. Despite the need for these studies, the FDA is proceeding prematurely with inserts on selected products. We think the Congress is the only place where the matter can be given the proper legal status and direction, particularly since it represents a conceptual change in the legal, medical and social framework of the nation's prescription drug information system.

The Solution

The PMA believes that carefully-devised pilot studies of various kinds of inserts are needed. They should be developed and implemented with full participation by doctors, pharmacists, consumers, communications experts and the drug industry. Such studies will provide reliable pathways to follow, so that inserts will be useful aids to medical practice.

And particularly we think that you should be closely involved in this debate and in these studies and decisions. Otherwise, people with less experience and qualifications may control the purposes, content and use of a tool with considerable promise for improved patient care. It could make a difference in your practice tomorrow, and more importantly, in the health of your patients.

PMA
THE PHARMACEUTICAL MANUFACTURERS ASSOCIATION
1155 FIFTEENTH ST., N. W., WASHINGTON, D. C. 20005



If you've been prescribing pentobarbital or secobarbital for insomnia, there's good reason to reconsider.

More effective than secobarbital through 14 nights of administration...[1,2]

In two separate sleep laboratory studies,[1] secobarbital 100 mg was found to lose much of its initial hypnotic effect in insomniac subjects within a two-week administration period. Dalmane® (flurazepam HCl), however, has been proved[2] to remain effective for both inducing and maintaining sleep at the end of two weeks, with the usual adult dosage (30 mg *h.s.*). Elderly and debilitated patients should receive 15 mg initially, to help preclude oversedation, dizziness or ataxia.

And more effective than pentobarbital through 28 nights of administration...[3,4]

In an original study designed to evaluate hypnotic effectiveness for 28 consecutive nights of use, the relative ineffectiveness of pentobarbital was established after only two weeks.[3] Dalmane, however, remained effective not only for 14 nights, but for 28 nights in chronic insomniacs,[3,4] without increasing dosage from night to night. Prolonged administration of Dalmane is seldom necessary, but when it is, periodic blood counts and liver and kidney-function tests should be performed.

More proven safety benefits for your patients than barbiturates...

Specific safety benefits not shared by barbiturate hypnotics: Dalmane (flurazepam HCl) may be used in patients on chronic warfarin therapy; no unacceptable fluctuation in prothrombin time has been reported.[5,6] And Dalmane has been proved not to interfere chemically with many common laboratory tests.[7-9] (Alterations have been reported due to pharmacological effects; see Adverse Reactions section of complete product information.)

Dalmane® (flurazepam HCl)

30-mg and 15-mg capsules

Unsurpassed record of efficacy and safety

Before prescribing Dalmane (flurazepam HCl), please consult complete product information, a summary of which follows:
Indications: Effective in all types of insomnia characterized by difficulty in falling asleep, frequent nocturnal awakenings and/or early morning awakening; in patients with recurring insomnia or poor sleeping habits; in acute or chronic medical situations requiring restful sleep. Since insomnia is often transient and intermittent, prolonged administration is generally not necessary or recommended.
Contraindications: Known hypersensitivity to flurazepam HCl.
Warnings: Caution patients about possible combined effects with alcohol and other CNS depressants. Caution against hazardous occupations requiring complete mental alertness (*e.g.*, operating machinery, driving).

Usage in Pregnancy: **Several studies of minor tranquilizers (chlordiazepoxide, diazepam, and meprobamate) suggest increased risk of congenital malformations during the first trimester of pregnancy. Dalmane, a benzodiazepine, has not been studied adequately to determine whether it may be associated with such an increased risk. Because use of these drugs is rarely a matter of urgency, their use during this period should almost always be avoided. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.
Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.
Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.*, excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.
Dosage: Individualize for maximum beneficial effect. *Adults:* 30 mg usual dosage; 15 mg may suffice in some patients. *Elderly or debilitated patients:* 15 mg initially until response is determined.
Supplied: Capsules containing 15 mg or 30 mg flurazepam HCl.

References:
1. Kales A, *et al*: *Clin Pharmacol Ther 20*: 541-545, Nov 1976
2. Kales A, *et al*: *Clin Pharmacol Ther 19*:576-583, May 1976
3. Kales A, *et al*: *Clin Pharmacol Ther 18*:356-363, Sep 1975
4. Dement WC, Guilleminault C, Zarcone V: Progress in clinical sleep research. Scientific exhibit at the American Medical Association, Atlantic City NJ, Jun 14-18, 1975
5. Robinson DS, Amidon EL: Interaction of benzodiazepines with warfarin in man, in *The Benzodiazepines*, edited by Garattini S, Mussini E, Randall LO. New York, Raven Press, 1973, p. 641
6. Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
7. Moore JD, Weissman L: *J Clin Pharmacol 16*:241-244, May-Jun 1976
8. Spiegel HE: Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
9. Kaplan SA, *et al*: *J Pharm Sci 62*:1932-1935, 1973

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80 mg trimethoprim and 400 mg sulfamethoxazole

OLD SIZE: NEW SIZE:

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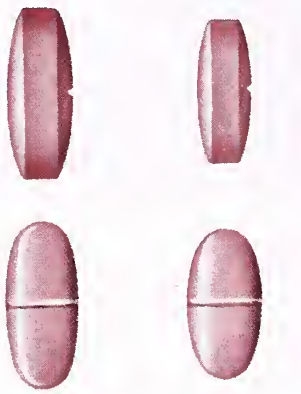
Each tablet contains:
160 mg trimethoprim and 800 mg sulfamethoxazole

OLD SIZE: NEW SIZE:



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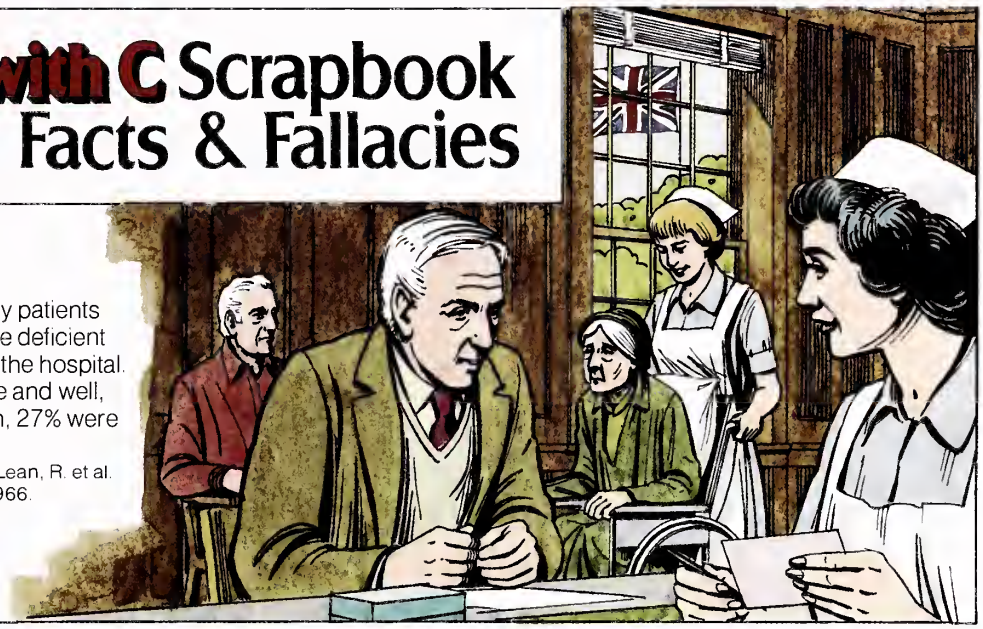

A study conducted among elderly patients in England showed that 41% were deficient in ascorbic acid on admission to the hospital. Even among those living at home and well, or not sufficiently ill for admission, 27% were deficient in ascorbic acid.

Griffiths, L.L., Brocklehurst, J.C., MacLean, R. et al. *Diet in Old Age*. Brit. Med. J., 1:739, 1966.

The loss of riboflavin in milk in a glass container exposed to sunlight for two hours may be as high as 95%.

Quick freezing of vegetables is accompanied by very little ascorbic acid loss. But blanching, washing, and prolonged standing at room temperatures results in considerable reduction in Vitamin C content.

In World War I a unit of 100 beds per division in the Russian army was set aside for scurvy patients. Yet, only 20 cases of scurvy were reported among all American troops in 1917-18.

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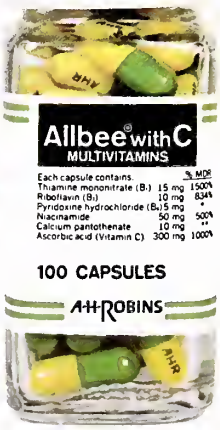

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Spasm reactor?

Donnatal®!

	each tablet, capsule or 5 ml tsp of elixir (23% alcohol)	each Donnatal No. 2 Tablet
Phenobarbital (warning: may be habit forming)	(¼ gr) 16.2 mg	(½ gr) 32.4 mg
Hyoscyamine sulfate	0.1037 mg	0.1037 mg
Atropine sulfate	0.0194 mg	0.0194 mg
Hyoscine hydrobromide	0.0065 mg	0.0065 mg

Indications: Based on a review of this drug by the NAS/NRC and/or other information, FDA has classified the following indications as possibly effective: adjunctive therapy in the treatment of peptic ulcer; the treatment of the irritable bowel syndrome (irritable colon, spastic colon, mucous colitis) and acute enterocolitis. Final classification of the less-than-effective indications requires further investigation.

Brief summary. Contraindicated in patients with glaucoma, renal or hepatic disease, obstructive uropathy (for example, bladder neck obstruction due to prostatic hypertrophy) or a hypersensitivity to any of the ingredients. Blurred vision, dry mouth, difficult urination, and flushing or dryness of the skin may occur at higher dosage levels, rarely at the usual dosage.

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in Brief

A summary of AMA, medical & health news

American Medical Association / 535 North Dearborn Street / Chicago, Illinois 60610 / Phone (312) 751-6000 / TWX 910-221-0300

AMA's new section on medical schools elected its first chairman and first delegate during the 1977 Annual Convention. Steven C. Beering, MD, dean, U. of Indiana School of Medicine, was named chairman, and Robert Stone, MD, dean, U. of Oregon School of Medicine, was elected delegate to the AMA's House of Delegates.

Physicians in undesignated PSRO areas were urged by the AMA House of Delegates recently to review decisions on establishing physician-controlled PSROs. After Jan. 1, the HEW secretary will be authorized to name qualified alternate organizations as PSROs in areas where one is not sponsored by physicians. Such areas exist in eight states. The House also approved criteria for alternate PSROs and called on the AMA to urge their adoption by HEW. AMA-developed legislation has been introduced in Congress to extend the alternate PSRO deadline by one year.

The AMA had submitted 56 testimonies, statements or letters, as of July 1, to Congress and federal administrative agencies on various topics.

The right of housestaff to conduct collective bargaining at the U. of Nebraska Medical Center was upheld last month in a 6-1 decision by the Nebraska Supreme Court. The court rejected the decision of the National Labor Relations Board last year that housestaff are students and should not have contract bargaining privileges. At the request of the House Officers Assn. of the medical center, the AMA submitted a "friend of the court" brief in the case. AMA policy states that housestaff are considered both students and employees, and should have the same right to collective bargaining as other employees.

A nationwide print advertising campaign, utilizing state, county and specialty medical society journals and bulletins, has been launched by the AMA Dept. of Membership Development. The campaign's purpose is to promote membership and to publicize AMA services and benefits available to the physician member. The ad slogan is "Join Us. We Can Do Much More Together."

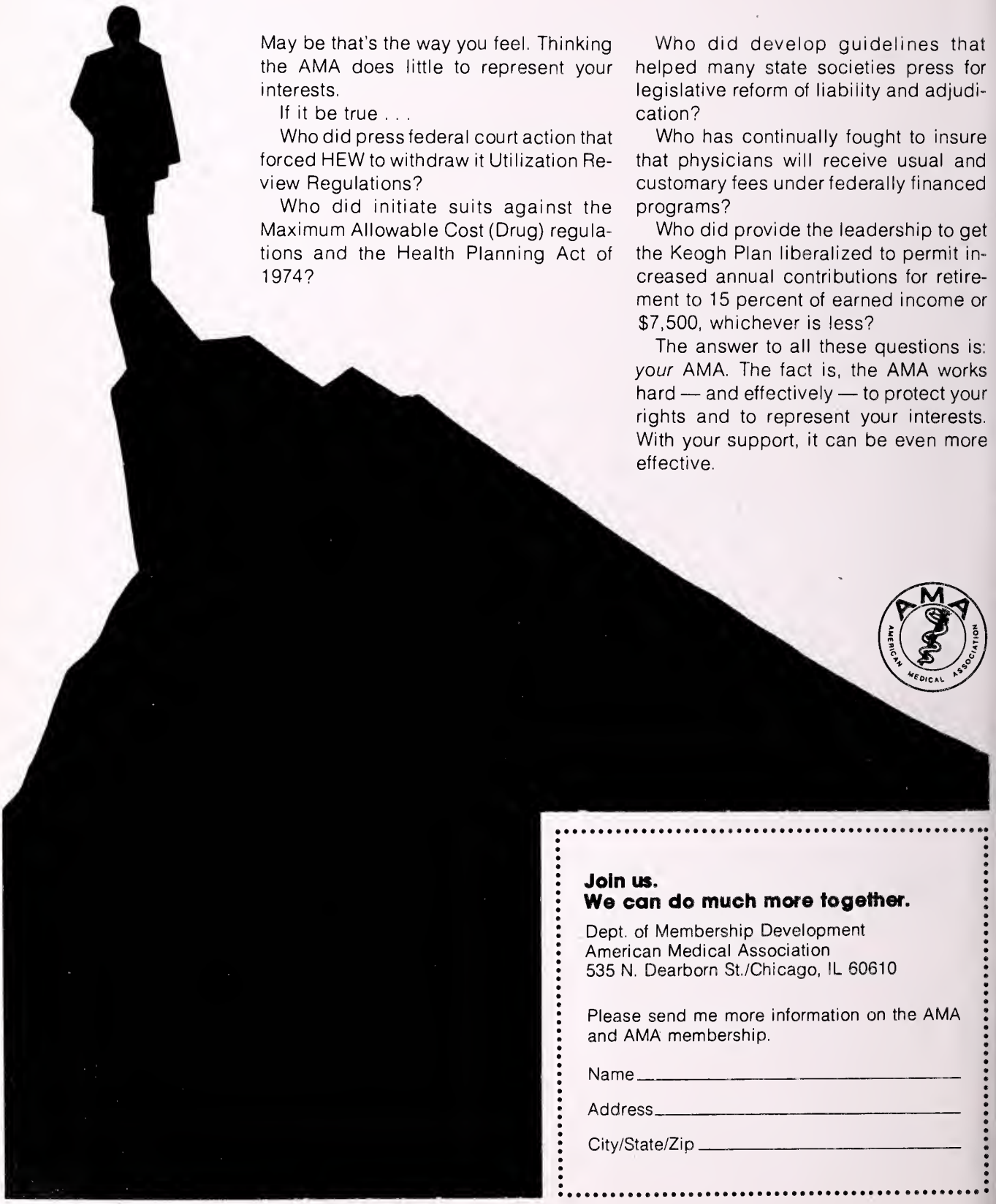
The average net income of physicians was $51,997 in 1974, the AMA told Congress in a letter listing fees by specialty from 1970-74. Sen. Herman Talmadge (D-Ga.) had asked for the information when the AMA testified on the Medicare-Medicaid Administrative and Reimbursement Reform Act. The average net income was $41,789 in 1970, $45,278 in 1971, $47,240 in 1972, and $48,574 in 1973. The AMA pointed out in its letter that "variations in net income and expenses among specialties and geographical regions cannot be explained on the basis of simple generalizations . . . the demand for varying services and additional independent factors must be considered." Figures were taken from the 1977 edition of **Profile of Medical Practice.** "Profile" and its campanion book, **Reference Data on the Socioeconomic Issues of Health** are prepared by the AMA's Center for Health Services Research and Development. Both books are available from the Order Dept., AMA Headquarters. The cost for each book is $4.50.

The U.S. Office of Education granted the Liaison Committee on Medical Education recognition as the accrediting agency for medical schools for a two-year period. LCME's eligibility had been challenged in March by the Federal Trade Comission's Bureau of Competition on grounds of "potential" conflict of interest.

A check for $1,512,566 was presented by the AMA Auxiliary to the AMA Education and Research Foundation during this year's Annual Convention. The Auxiliary's gift set a record for contributions to AMA-ERF.

First fellow in the AMA's Dr. Morris Fishbein Fellowship in Medical Journalism is Jeffrey R.M. Kunz, MD, a recent graduate of the U. of Wisconsin Medical School and former editor of the medical school newspaper. Dr. Kunz will spend 10-months at AMA Headquarters, working in all aspects of professional publishing. The fellowship was established last year by the Board of Trustees in memory of the late Morris Fishbein, MD, former editor of **JAMA.**

Quote:
The AMA doesn't represent me.
Unquote.



May be that's the way you feel. Thinking the AMA does little to represent your interests.

If it be true . . .

Who did press federal court action that forced HEW to withdraw it Utilization Review Regulations?

Who did initiate suits against the Maximum Allowable Cost (Drug) regulations and the Health Planning Act of 1974?

Who did develop guidelines that helped many state societies press for legislative reform of liability and adjudication?

Who has continually fought to insure that physicians will receive usual and customary fees under federally financed programs?

Who did provide the leadership to get the Keogh Plan liberalized to permit increased annual contributions for retirement to 15 percent of earned income or $7,500, whichever is less?

The answer to all these questions is: *your* AMA. The fact is, the AMA works hard — and effectively — to protect your rights and to represent your interests. With your support, it can be even more effective.

Join us.
We can do much more together.

Dept. of Membership Development
American Medical Association
535 N. Dearborn St./Chicago, IL 60610

Please send me more information on the AMA and AMA membership.

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If the Decision Were by the Sick

The increase in cost of medical care is consistent with increase in cost of most other major parts of our economy - food, gasoline, and housing, etc. But physicians know that this is not the real issue. The fact is, the cost of medical care should not be addressed the same as other costs. There are some things that cannot be measured in dollars - as we learned at our mother's knee.

Take the hallmark of the present debate on cost containment - the CT Scanner. What is the cost of two or three days' wait, or longer, for examination by one of the limited numbers? It is not only the hospital per diem!. It is not only the occasional cost in life where immediate examination could have been lifesaving. An immeasurable cost of delay is the worry and agonizing of the patient until the diagnosis is made.

Every patient wants the best. But this fact is not the prime concern of the health planner who makes his decision by numbers and statistics. This fact is not appreciated by the bureaucrat of HEW whose personal health care is guaranteed This is not a worry of Senator Kennedy and the authorship of NHI whose financial position has always assured that there be no wait for the best there is.

Perhaps the decisions on cost of medical care should be made only by those who have been seriously ill. Such decisions would be to the advantage and deep appreciation of those who are yet to be sick.

Dr. Kenneth Sawyer — Memoriam

In our Rocky Mountain region, Dr. Kenneth Sawyer has been a leader in medical activities for many years. In his years on the Board of Trustees of the American Medical Association he represented our area with energy, wisdom, and devotion. A recent President's Letter in *Colorado Medicine* is appropriately presented in this journal.

Early Friday morning, July 29, 1977, one of the great leaders of the Colorado Medical Society did not waken for his usual six-thirty arrival at his hospital. Dr. Kenneth Sawyer had finished a medical career begun in 1931 when he graduated from the University of Colorado Medical School. Into those forty-six years he had crowded many times more activity than his surgical practice, the biggest in town. A list of Dr. Sawyer's activity and accomplishment in his city and state government, his university and medical school, the top professional societies of his specialty, the Denver Medical Society, the Colorado Medical Society and the American Medical Association would fill all the pages of this bulletin. Dr. Sawyer was always involved in more action in more places than seemed possible to the rest of us, and yet he was doing the ordinary everyday things along with us. He was always among the leadership in these activities, and yet he was always right there in the trenches helping others along, especially the young doctor on the way up. He was always helping someone. No matter how you tried, you were always in debt to Dr. Sawyer—and always will be. That's the way he wanted it.

Kenneth Sawyer believed in the system. In his full life of involvement, public and professional, he had confidence in democracy. In local or national government, in the medical society or the American Medical Association, he had faith in the democratic process. He was more aware than most of us that a democracy *MOVES SLOWLY*. But he had great patience with its workings, because he was confident that it is the system most certain to *MOVE CORRECTLY*. He never had time to criticize, he was busy becoming involved to make a change. He recognized the strength and talents of his associates and was patient and tolerant of their weaknesses—that's why he was a leader.

Kenneth Sawyer was happiest when he was helping someone and was hard at work at several jobs. I hope God has a lot of things He needs done.

Second Class Medicine

Recently in the Provincetown, Massachusetts *Advocate* a physician detailed one doctor's difficulties with the Medicaid system in his practice in Paterson, New Jersey. The good and the bad of Medicaid, including the well publicized scandals, are familiar to many. Not so well known is the thwartening of legislative purpose by administrative bureaucracy - *not unexpected to those aware of other attempts to establish social or economic equality by government fiat.* We are indebted to the A.M.A. News Release of August 3, 1977 for the following account by Robert A. Goldstone, MD, entitled "How the U.S. Turns Medicaid into Second Class Medicine."

A cruel hoax has been perpetrated by the government upon the recipient of Medicaid. The Medicaid bureaucracy is doing its best to see that there will continue to be two levels of medical care, and that the Medicaid patient will remain a second class patient.

By way of historical background, the intent of the Medicaid law was to remove the medically indigent patient from the clinic setting, to abolish his status as a "charity case," and to provide for him (or her) the same quality of medical care that the paying public was assumed to be receiving. This was the promise of the program.

From the outset, the performance was hampered by restrictive regulations which, contrary to the intent of the program, made it impossible for a physician to treat a Medicaid patient in the same fashion that he would treat a private patient. For example, since Medicaid would not reimburse him for the cost of an expensive medication which he might inject, or for a device or appliance which he customarily dispensed, he was faced with the choice of either altering the method of dealing with that patient or his problem, or suffering a net loss with respect to reimbursement for that patient visit, if the cost of the materials supplied exceeded the allowance paid by Medicaid.

That allowance in itself was far below the usual and customary fee the physician had been charging, which has been recognized by Medicare and other insurance agencies.

When the physician is prevented from choosing the same treatment for his Medicaid patients as he selects for his other patients, the government has in fact established a "second class" medical system for the indigent. The patient is therefore thrust once more into the role of receiving "charity care" from the physician.

In addition, the program has become a bureaucratic nightmare. The paperwork is impossible. Every judgment is questioned and criticized, frequently by people with little or no medical knowledge. Hospitalized patients are suddenly denied benefits, and access to the appeals system is restrictive and time-consuming.

We have all read of "Medicaid Mills." Why do they exist? Because only by processing large numbers of patients and ordering large numbers of laboratory tests, and otherwise taking advantage of the loopholes and weaknesses in the Medicaid law is it possible to afford to treat other than an occasional Medicaid patient. A physician who lives in a community with a significant number of Medicaid patients, and who wishes to practice good, ethical medicine, finds it difficult to do so at the Medicaid reimbursement rate.

The public must be aware that a medical office can have a tremendous overhead. Only the work of the physician in the office produces income, yet there are one to four nurses, assistants, technicians, and receptionists, increasing rents, increasing heating and electrical bills, sky-rocketing insurance premiums for malpractice, and a host of other expenses which the patient generally does not stop to think about. The individuals to whom these bills are owed do not extend "charity" and are not concerned about whether or not the physician is treating Medicaid patients.

Recognizing that a crisis does exist, I would hope that some of the anger expressed at the medical profession, could be directed at the governmental agency which has ordered that care be provided, but failed to provide the necessary funds to pay for that care.

I am sure that if the government promised poor and hungry people free milk, and arranged for it to be provided by dairies who would be reimbursed at the rate of $1 a gallon, well below the market value, there would be no dairies providing milk. The government has done the same thing with Medicaid.

I have hesitated thus far to touch upon the even more sensitive aspect of a Medicaid practice. It would be easy to misconstrue my remarks as prejudicial, which they are not meant to be. For a variety of societal and economic reasons, it has been the experience in my practice that the following problems exist more frequently with Medicaid patients than with the balance of my patient population:

1) There is a very high percentage of patients who seek medical attention for minor complaints and for problems that are self-limited and require no medical attention, for which paying patients would not think of calling a physician, and which do not require the attention of a physician.

2) There is a significant higher percentage of "no-shows" in the Medicaid population than in the general population. A no-show represents an intolerable loss of productive time for a physician who is trying to work by a reasonable schedule.

3) The Medicaid patient tends to bring with him more people, (including children who remain unsupervised during their parent's examination, disrupting the office routine and tranquillity), who utilize a disproportionate amount of the available waiting room space so that there is insufficient room for the remaining patients and visitors.

4) There is a greater tendency not to follow the recommendations of the physician, which is a frustrating experience for the doctor, and which hampers rational delivery of health services.

Now I am certain that those who read this hastily will conclude that I am a bigot, against the poor, and a very prejudiced person.

Personally, I believe that my liberal credentials are in order, and I would point out that not only do I practice in Paterson, N.J. which is overwhelmingly populated by minority groups, but also that I am about to move my office further into the heart of the downtown area.

It is necessary to point out that there is a tremendous problem involved in attemptintg to deliver high-quality medical care to patients who have never experienced it before, who hold different values, who are used to being treated like numbers instead of individuals, who are unable to find babysitters to watch their families when only one needs to be examined, and who are unable, by training and experience, to make even slightly sophisticated judgments regarding the necessity for obtaining health care and for following orders.

Couple these patients and problems in a significant percentage of the Medicaid population with a repressive system of reimbursement, which in fact can result in a payment to the physician of less than the actual cost of goods and services rendered to the patient, and it should be understanda-

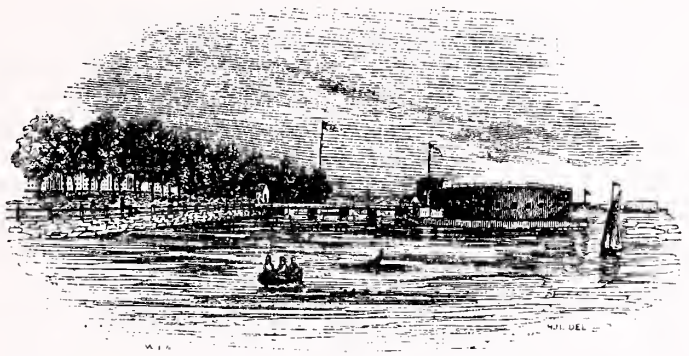
ble why increasing numbers of physicians find it difficult to participate in the program.

By way of example, let me point out that in my locality, a specialist is reimbursed $7.56 for an office visit. If he provides an elastic bandage, crutches, or utlilizes supplies for dressings, he is allowed no more money. The absurdity of this situation, is apparent when the patient is referred instead to the hospital clinic. Reimbursement to the clinic is based upon a formula which takes into account the cost of providing care to that patient. Followup visits at the clinic at the hospital where I practice, which is a Catholic non-profit hospital, are reimbursed at the rate of $35 per visit—and this figure is arrived at by taking into account actual costs.

If a non-profit institution finds that it costs $35 to examine and treat a patient in a clinic setting, how can the government justify payments of less than one quarter of that amount to the physician who renders personal care, in dignified surroundings, to that same patient with the same problem and with the same needs?

The answer is, that it can't, but it finds it impossible to underpay hospitals, while they can expect paying patients to make up the difference in the doctor's office. When the saturation point is reached, that is, when the physician can no longer pass on his costs or absorb them, he is faced with the choice of either reducing his Medicaid practice (and these patients then return to the clinics at a four fold *increased* cost to the government!), or compromising his standards of care and creating a second level to comply with Medicaid imposed restrictions.

I believe that the Medicaid patient is entitled to the same "first class" medical care as the rest of my patients. If the government holds out that promise to the indigent, it must be prepared to back it up. I join with all thoughtful people who condemn the abusers of the system, and ask that we all join in an effort to urge the government to show that it intends to make it work, and fulfill its promise, which until now has been a hollow one.



The Battery, and Castle Garden.

Social Security Disability and the Practicing Physician

A medically determinable impairment which prevents the individual from engaging in substantial gainful employment for twelve months or more is a prerequisite for those applying for Social Security disability benefits.

Documentation of medical evidence by a treating physician or by a physician to whom the claimant has been referred for consultative examination must be secured by the Disability Determination Services.

Such examinations require a detailed history and physical as well laboratory work, x-rays, EKG's, pulmonary function studies, etc, with special emphasis on the objective findings. The weight of evidence in determining the allowal or denials of the claimant is predicated on the objective findings if it is not possible to verify subjective statements by the claimant on the basis of muscle spasms, range of motion restrictions, abnormal x-rays, or decreased pulmonary functions.

It is not always clear to a claimant that even when an impairment prevents the individual from continuing in an established job and this individual can function in another capacity that benefits are denied, unless the option is taken.

Evidence from the practicing physician either as treating physician or one called in consultation is crucial, and the more detailed the reports, the more equitable is the final decision. The Disability Determination Services personnel wishes to express its appreciation for the cooperation of the medical profession.

The fee schedule is to be reviewed on a yearly basis according to present plans, so that proper adjustments to fees that are far behind the cost of inflation can be made.

Familial antithrombin III deficiency

*An Important Clinical Entity**

Martin J. Rubinowitz, MD, Denver, and Clyde Ford, MD, Salt Lake City, Utah

Familial antithrombin III deficiency is an entity which is not well known, easily diagnosed, and should be suspected in patients with recurrent thromboembolic phenomena. Long-term anticoagulant therapy may be lifesaving.

Antithrombin III (AT-III) is a circulating protease inhibitor believed to derive its major physiologic importance as an inhibitor of thrombin. Although a laboratory finding initially, it has recently assumed increasing clinical importance with the description of several families demonstrating a genetic predisposition.[1-4]

Afflicted individuals suffer recurrent and occasionally fatal thromboembolic phenomena. It is likely that AT-III deficiency is far more common than is generally realized by practicing physicians. Indeed, it has been estimated that as many as two percent of clinical pulmonary emboli may be due to this etiology.[5] Thus, many cases are missed, an unfortunate circumstance, as the diagnosis can be made on the basis of a relatively simple coagulation test, and treatment with long-term and perhaps lifetime anticoagulation may be lifesaving.

Our study of a new AT-III deficiency kindred has re-emphasized to us the clinical importance of this illness. It is the purpose of this paper to review the clinical aspects of AT-III deficiency, encourage a higher index of suspicion among practicing physicians, and stimulate more widespread availability and use of screening tests.

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Material and Methods

Family members were screened by two methods. The coagulation method of Von Kaulla[6] consists of adding thrombin to the patients's serum and waiting six minutes. Then the mixture is added to a fibrinogen solution and the time needed for a clot to form is determined. Normal values are 22-125 seconds.

The second method is an immunologic technic devised by Mancini[7] using radial immunodiffusion plates manufactured by Behring Diagnostics. The patient's serum containing antithrombin is added to the well and diffuses into an anti-antithrombin media. The distance from the well that the precipitin line forms determines the antithrombin III concentration.

CASE REPORT

A 32-year-old male was admitted to St. Joseph Hospital, Denver, on September 15, 1975. One week prior to admission he developed low back pain for which he took large amounts of aspirin and codeine. Two days prior to admission he developed hematemesis and was hospitalized in Laramie, Wyoming. He was given plasma and four units of blood prior to transfer. He had a history of two episodes of lower extremity thrombophlebitis for which he was heparinized in the past. He had an impressive family history of thromboembolic disease.

On admission he was awake but clammy and cold, complaining of low back and abdominal pain. His blood pressure was 134/80 supine and 90 systolic in the sitting position. His pulse was 140 and regular. Abdominal examination showed distension and hypoactive bowel sounds without evidence of peritoneal irritation. Lower extremity examination showed stasis dermatitis without evidence of active thrombophlebitis.

Admission laboratory data included an hematocrit of 38 per cent, white blood count of 20,800 with a left shift, and a platelet count of 92,000 (normal 150-300,000). Radiographic workup included an abdominal arteriogram which showed evidence of a pancreatic mass (? pancreatitis) but no specific bleeding point. Upper G.I., small bowel series and barium enema demonstrated esophageal varices and nearly complete obstruction at the ligament of Treitz with an abnormal segment of proximal jejunum, suggesting edema or infiltration of the bowel wall and raising the possibility of infarction. A sonogram showed an enlarged pancreas and splenomegaly, and a liver-spleen scan revealed a normal liver but no splenic uptake consistent with splenic vessel thrombosis. Endoscopic workup identified bleeding from above the level of the proctoscope, a small esophageal vein and minimal gastritis.

Abnormal preoperative clotting studies included thrombocytopenia, slightly elevated fibrin split products, and a few Schistocytes on smear. Because of suspected disseminated intravascular coagulation, the patient was heparinized.

On September 23rd he was subjected to an exploratory laparotomy and total thromboses of the portal, splenic and mesenteric veins were found. The gangrenous bowel was resected and splenectomy was performed.

I (MJR) saw the patient in consultation on September 24, 1975, three days postoperatively, at which time he had developed acute renal failure and was being hemodialized. AT-III deficiency was suspected and confirmed by an extremely low value of 7 seconds (normal 22-125 seconds). Since heparin may have been contributing to this low value, his mother, who was asymptomatic, was checked and had a value of 8 seconds. His heparin doses were increased but were extremely difficult to determine because variable amounts of heparin were needed to keep open his A-V shunt, which frequently clotted. Coumadin could not be administered because of the absence of an oral route.

His subsequent course was downhill, marked by numerous complications including a wound infection, persistent renal failure, gastrointestinal bleeding and liver failure. He expired on October 12, 1975.

Other members of the family have been studied by both the clotting and immunologic technics detailed previously. There was usually close agreement between the two methods. To date, sixteen members have been discovered to have AT-III deficiency.

TABLE 1

Antithrombin III Levels

Decreased	Increased
Hepatitis	Coumadin
Cirrhosis	Progesterone
Estrogens	Hemophilia
Pregnancy	Proconvertin deficiency
Postoperatively	Uremia
Heparin	Congenital or acquired heart disease without CPC
Post-myocardial infarction	Androgens

Discussion

At-III deficiency has been considered a rare disease because of relatively few reports to date. More likely, there exists a large number of undiscovered families. It is possible that two per cent or more of patients with venous thromboembolism have AT-III deficiency.[5]

The antithrombin III molecule is an alpha II globulin with a molecular weight of about 64,000.[8] It appears to be synthesized mainly in the liver and its major action is to inhibit activated enzymes within the clotting sequence which include, in addition to thrombin, activated factors IX, X, XI and XII. Whether the clinical deficiency is due to decreased synthesis of normal molecules or production of abnormally functioning ones has not been determined.[4]

At-III deficiency is inherited as an autosomal dominant with incomplete penetrance. The disorder is characterized by an increased risk of thromboembolic episodes usually manifest as lower extremity thrombophlebitis and pulmonary embolism. The likelihood of these clinical phenomenon occurring appears to correlate with the level of deficiency. Affected individuals often have levels of 50 to 60 per cent of normal and experience the onset of clinical manifestations after age 16.[4] Some family members have thrombotic episodes during high risk periods such as pregnancy, in the postpartum period, when taking contraceptives and with surgery or other trauma.

The diagnosis can be made by demonstrating a low inhibitor activity with the coagulation method of Von Kaulla[6] or by direct measurement of AT-III levels with the commercially available radioimmuno-diffusion assay.[7] Both tests are relatively inexpensive, reliable and rapid. A low level, however, is not necessarily diagnostic of AT-III deficiency as several clinical states may be associated with decreased AT-III (See Table 1). These include disseminated intravascular coagulation, a variety of liver diseases,[1] exogenous estrogen intake, and surgery.

AT-III levels are usually depressed by oral estrogen-containing contraceptives in the first cycle and may return to normal approximately 18 days after they are discontinued. Whether this effect is dose related is controversial.[8 9] Values of less than 15 seconds have been as-

sociated with clinical thromboembolic episodes.[10] Discontinuation of estrogens resulted in either complete remission or significant improvement in eleven of twelve patients studied by Zuck et al.[10] These authors suggested that an AT-III level less than 15 seconds is a contraindication to the continued use of estrogens. Likewise, estrogens may also be contraindicated in the post-partum period for suppression of lactation because reduced AT-III levels persist much longer than in non-suppressed patients.[11]

Following surgery, AT-III levels are depressed reaching a nadir three days postoperatively.[12] Heparin may accentuate this reduction, possibly by increasing AT-III utilization. It has been shown in two studies that women on oral contraceptives and patients undergoing total hip surgery who had low AT-III values preoperatively were more likely to develop deep vein thrombosis postoperatively.[13] Perioperative anticoagulation would seem indicated in such patients.

Both heparin and Coumadin have been used for the treatment of AT-III deficiency. Because most studies have shown that AT-III is the substance through which heparin exerts its anticoagulant effect,[1,14] the use of this agent in patients who have AT-III deficiency might be questioned on theoretical grounds. In clinical trials, however, Von Kaulla felt that heparin was the drug choice. This drug successfully prevented clotting episodes in all of his patients. On the other hand Coumadin, possibly by stimulating the synthesis of AT-III, usually elevated the level to the normal range, often within a few days.[1] Von Kaulla found that Coumadin did not protect those patients from further thrombotic episodes; in these patients AT-III levels did not rise even when the prothrombin time was in the therapeutic range. They were protected if AT-III levels rose to normal, even though the rise was often delayed.[4]

Gruenberg[15] reported a patient similar to our propositus who developed mesenteric venous thrombosis. He recommended prophylactic therapy be used for asymptomatic individuals with AT-III deficiency at times when the risk of thrombosis is high, such as during elective surgery, postpartum, etc. He felt that heparin was unreliable, that Coumadin therapy was encouraging, and stated that AT-III concentrates are being developed. He also emphasized that AT-III deficiency is missed by the usual coagulation screening workup.

In searching for a good screening test one looks for a procedure which is inexpensive, rapid and reproducible. The AT-III level fulfills these criteria. A clotting assay such as Von Kaulla's method costs approximately $15.00 and takes about three hours to perform. The serum can be stored at -15° centigrade for a few hours. The results may vary 10-20 per cent with storage. The immunoassay is less expensive, simpler to perform and serum can be stored at -40° centigrade for up to three weeks, so that samples can be shipped in dry ice.

A logical sequential approach is as follows: if the patient has a history of recurrent thromboembolic episodes, especially if he has a positive family history, an AT-III level should be drawn. If it is low, available family members should be checked. If the diagnosis is confirmed, heparin and coumadin should be administered simultaneously if the patient has an acute thromboembolic episode or if he is asymptomatic but at high risk for thrombosis (i.e. during surgery, etc.). For patients over the acute episode or who have previous history of thromboembolic episodes, Coumadin should be administered. If the AT-III level rises to normal, the patient should be continued on this medication on a longterm and perhaps a life time basis. If the AT-III levels do not rise or subsequent thromboembolic episodes occur, then longterm subcutaneous heparin therapy should be given strong consideration. If the patient is actively bleeding, fresh whole blood or fresh frozen plasma can be administered if AT-III concentrates are not available. Family members in whom AT-III deficiency is identified should be advised to be similarly evaluated and treated.

It is most important that AT-levels be drawn in women with a family history of thrombosis before prescribing estrogens. We would suggest avoiding or discontinuing estrogens in any patient whose AT-III levels are borderline or low. Low dose progestational agents were associated with an elevated AT-III level and felt to be safe for contraception.[16] Androgens have also been demonstrated to elevate AT-III levels.[17]

Summary

Familial AT-III deficiency is an important clinical entity which should be suspected in any patient with recurrent thromboembolic

episodes, especially with a positive family history of thromboembolism. AT-III determinations are rapid, inexpensive and accurate. One should be aware that other conditions may alter the level. Longterm anticoagulant therapy for symptomatic patients and short-term therapy for asymptomatic patients at times of increased risk is indicated and may be lifesaving. Avoidance of estrogens is recommended to any patient in whom AT-III levels are borderline or low. A sequential approach for diagnosis and treatment of this entity has been outlined.•

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Surgical complications of anticoagulants*

Paul R. Radway, MD, Pueblo, Colorado

Because of the increasing number of patients taking anticoagulants, review of the manifold complications is timely.

Anticoagulants are widely used in the treatment and prophylaxis of various thromboembolic disorders, and it is estimated that more than 1,000,000 Americans are currently on long-term anticoagulant therapy. The incidence of significant bleeding complications (defined as an episode requiring the discontinuance of the anticoagulant and/or blood transfusions) is difficult to assess, but is about one episode per ten patient-years. Approximately one-half of all significant complications are abdominal in nature. The key to management of these problems is to ascertain early that the patient is anticoagulated, thus enabling the clinician to suspect and search for a complication.

Non-Abdominal Complications

Hematuria and epistaxis are the most common bleeding episodes in anticoagulated persons, and are usually of short duration and little significance. Serious or repeated occurrences demand a careful examination to look for an underlying cause, and should include intravenous pyelography and cystoscopy, if the problem is blood in the urine.

Hemorrhagic necrosis of the skin is probably a hypersensitivity phenomenon, rather than a bleeding complication. It has been seen only in patients on oral anticoagulants, usually 3-8 days after beginning therapy. Typically, it presents with the sudden appearance of a painful, tender, erythematous area of skin. This area demarcates, becomes black and blistered, and finally sloughs, unless it is first excised and grafted. Unfortunately, reversing the anticoagulant, administration of steroids, and other attempted remedies have not altered the course. Many cases have been reported of an entire breast sloughing from this process, but it can occur anyplace on the epidermis.

All other non-abdominal anticoagulant complications involve bleeding into a closed space, and the pathophysiology and therapy may be approached from the standpoint of pressure and bulk changes interfering with function. Intracranial hemorrhage is probably the most lethal complication of anticoagulants. Although the incidence of "strokes" in anticoagulated persons is no higher than in comparable groups without anticoagulation, the mortality is. Diagnosis is made in the usual manner, and arteriography is definitive. After reversal of anticoagulation, extra- or sub-dural hematomas should be evacuated. Intra-cerebral clots should be evacuated only if there has been some prior neurologic improvement, indicating reversible damage to the brain. Spinal cord hemorrhage should be considered in any anticoagulated patient who complains of backache or radicular pain with neurologic deficits. Myelography and clot evacuation should be undertaken, after reversal of the anticoagulant. Other nervous system complications include femoral nerve entrapment and carpal tunnel syndrome. Early decompression of these lesions is advised, to avoid late neurologic sequellae.

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Hemopericardium can occur, particularly in patients anticoagulated post myocardial infarction. Cardiac tamponade is diagnosed by noting distended neck veins, hypotension with a decreased pulse pressure, pulsus paradoxus, and faint heart tones on auscultation. Treatment is reversal of anticoagulation followed by pericardiocentesis. Constrictive pericarditis requiring pericardiectomy may be a late sequella. Hemothorax presents with chest pain, sometimes accompanied by shock and dyspnea. Fluid is seen on chest x-ray and the diagnosis is confirmed by thoracentesis (after reversing the medication). Closed-chest drainage is adequate treatment.

Hemarthrosis in anticoagulated persons is not as common as in hemophiliacs. Presentation is with a swollen, painful joint, and diagnosis is by joint aspiration, after reversal of the anticoagulant. Treatment is bedrest and joint aspiration. Intra-articular steroids may be of benefit. Other rare occurrences include bleeding into the posterior chamber of the eye, bleeding into a salivary gland, and hematoma of the larynx necessitating tracheostomy. Clearly, a bleeding complication must be suspected in any anticoagulated patient presenting with acute symptoms, so that vigorous and appropriate therapy can be undertaken.

Abdominal Complications

Gastrointestinal bleeding occurs in three to four per cent of all patients chronically anticoagulated. Though exsanguination is extremely rare, significant bleeding is not. A thorough search for an underlying lesion should be made. Treatment is reversal of the anticoagulant, supportive care, and specific treatment for any lesion found.

Abdominal complications may be classified as bleeding into the lumen, wall, or mesentery of the gut, retroperitoneal bleeding, and bleeding into or from other organs, including the abdominal wall. With the exception of GI bleeding, all other types frequently present with abdominal pain, nausea, and vomiting, distention, and abnormal bowel sounds. To sort out these patients with symptoms of an "acute abdomen" and begin the correct therapy is dependent upon a careful history and examination, and is often quite difficult.

Retroperitoneal bleeding should be considered when there is a dropping hematocrit without a known source of bleeding. Usually there is pain in the abdomen, back, or flank, with abdominal distention and decreased or absent bowel sounds. Occasionally there is a mass palpable in the flank, and ecchymosis may appear later. X-rays typically show a nonspecific ileus, and a mass obscuring the psoas or kidney shadows may be seen. Standard treatment is conservative, in which ureteral catheters are rarely used to relieve obstruction.

Intramural hematoma nearly always involves jejunum or ileum. The hemorrhage begins submucosally and dissects to give mucosal obstruction or bleeding. Dissection may also occur along the serosa into the mesentery and even into the retroperitoneal space. These patients present with abdominal pain, often cramping in nature, distention, nausea and vomiting, and sometimes obstipation. Examination reveals a distended, diffusely tender abdomen, with bowel sounds varying from increased to absent, depending on the state of the pathology. It is possible to make the diagnosis with a barium upper gastrointestinal examination, which usually shows a rigid narrowed segment with coarsened mucosal folds giving a "coiled spring" appearance. Treatment of choice is non-operative. Reversal of anticoagulation, IV fluids, and nasogastric suction will usually provide improvement within 48 hours. Serial upper GI x-rays have shown that the bowel will return to normal in 14 to 90 days.

Bleeding into an ovarian cyst, with rupture, may lead to rapid massive hemorrhage. "Spontaneous" rupture of the spleen or the kidney may also cause dramatic bleeding. Patients who present in shock, demand abdominal exploration, and appropriate treatment becomes obvious. Cases of bleeding in the vicinity of the pancreas have been described as pancreatitis, but documentation of elevated pancreatic enzymes is lacking, and this is probably another form of retroperitoneal hematoma. Several cases of hematocele of the gallbladder have been seen in patients with cholelithiasis and cholecystitis; diagnosis and treatment is similar to that in patients not anticoagulated.

Bilateral adrenal hemorrhage is a well-documented complication, although no adequate explanation has been offered for this local destruction of tissue. Presentation again

simulates an "acute abdomen", with epigastric pain, nausea, vomiting, distention, and decreased bowel sounds. Usually, there is also weakness, fever, hypotension, and disorientation, which may lead to a mis-diagnosis of gram-negative sepsis. Acute hyponatremia and hyperkalemia may occur, and provides a clue to the correct diagnosis. All reported cases have survived at least two days, which allows time to do an eosinophil count and studies of steroid levels. A direct eosinophil count greater than 50/cubic mm. in an acutely ill patient strongly suggests adrenal insufficiency, and a trial of steroid therapy is appropriate.

Rectus hematoma may also masquerade as an "acute abdomen" and indeed, many patients reported in the literature underwent celiotomy for diagnosis. However, there is often a history of slight trauma, such as coughing or straining, and the pain is usually persistent, increasing, and unilateral. A paralytic ileus is often present. The key to diagnosis is locating the tender abdominal mass which does not change on having the patient lift his head, indicating its presence in the abdominal wall. Treatment is reversal of the anticoagulant. It is not necessary to drain these lesions, although this can be done under local anesthesia if relief of symptoms is desired.

Approach in Abdominal Complications

Since the majority of anticoagulated patients are poor operative risks, it is desirable to avoid surgical intervention. The differential diagnosis must include all the causes of an "acute abdomen", particularly mesenteric vascular occlusion, intestinal obstruction due to causes other than anticoagulant complications, pancreatitis, cholecystitis, and appendicitis. Nevertheless, acute abdominal symptoms in an anticoagulated patient should force the clinician to consider the mentioned complications as leading possibilities.

A careful history and physical examination will often provide clues to the particular diagnosis. The abdominal wall mass characterizing rectus hematoma should be searched for specifically. Unless another diagnosis is clear, a direct eosinophil count should be done, in search of adrenal insufficiency. If borderline, serum steroid levels may be drawn and a therapeutic trial of corticosteroids initiated. X-ray films of the abdomen may show loss of the psoas shadows, pointing towards retroperitoneal bleeding as the diagnosis, or they may suggest intestinal obstruction. If the latter is found, barium x-ray examination of the intestine is in order, and may confirm a diagnosis of intramural hematoma. Paracentesis is unnecessary, and may be misleading, as free blood is often present in all of these situations (except rectus hematoma).

A therapeutic trial of nasogastric drainage, intravenous fluids and careful observation is often indicated. Evidence of continued rapid bleeding will demand exploration. Deterioration of the patient despite adequate nonoperative therapy will also require celiotomy. Occasionally, exploration of the abdomen will be necessary for diagnostic purposes only. It should also be remembered that one patient may have two diseases—diagnosis of an anticoagulant complication does not exclude a second condition requiring surgery.

If exploratory celiotomy is undertaken, and a bleeding ovarian cyst or spleen is found, the treatment will be clear. If a retroperitoneal hematoma is found, it is best to ascertain that the kidneys are intact, and nothing else; minimal disruption of the retroperitoneal space will allow tamponade of the bleeding site. An intramural hematoma will look at first like a segmental infarction of the bowel, but careful examination will show that the blood supply is normal, and no resection should be done. Deaths in patients with intramural hematomas have been reported only in those undergoing operation, and are often due to myocardial infarction. It is best not to operate on these patients, but if this lesion is found at surgery, minimal surgical trauma and operative time is indicated.

Occult Lesions

It is impossible to accurately estimate the incidence of otherwise occult lesions that are found because of bleeding while anticoagulated. Cancers revealed in this manner include renal, bronchogenic, gastric, colonic, and uterine. Benign lesions that have been found include nasal polyps, peptic ulcers, esophagitis, diverticuli and hemorrhoids. There is an increased yield in patients who bleed while clotting studies are within accepted "therapeutic range", versus those who are over-anticoagulated at the time of

the bleeding episode. Nonetheless, it is recommended that any patient who has a significant bleeding episode undergo a thorough search for an underlying lesion.

Summary

Complications of anticoagulant therapy are fairly common and often serious. A specific diagnosis is often difficult to make, but the key determination is to ascertain that the patient is anticoagulated, after which one can suspect a complication. The anticoagulant should always be discontinued, and administration of reversing agents is sometimes indicated. A thorough search for an underlying lesion should be made. Caution is recommended regarding surgery, but intervention will sometimes be necessary to determine the diagnosis or to remove damaged tissue and evacuate clots.●

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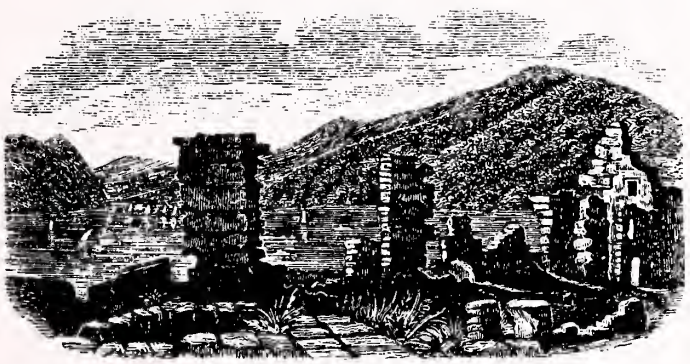

Ruins of Fort Ticonderoga.

Drug ototoxicity *

Dennis A. Greene, MD, Denver

The incidence of iatrogenic ear disease is low. Understanding the pharmacology and dynamics of new drugs and their effects on the delicate ultra structure of the ear has prevented many irreversible and tragic consequences of therapy. However, a "trade off" of sorts still exists for some patients who find that deafness is part of the price for cure of overwhelming sepsis or renal failure. The actual incidence is not known. Pre-treatment audiograms are usually not done. Many patients have been exposed to a variety of potentially ototoxic medications. Reactions may be subclinical; they may be transient; they may be unilateral or bilateral. Fairly reliable incidence figures are available only for the antibiotic gentamicin - approximately two per cent of patients develop vestibular reactions and approximately one per cent develop measurable hearing loss.

Drug effects on the ear involve one or both of two different systems - the auditory and the vestibular. Those affecting hearing are called cochleotoxic, because histologic changes may be found in the cochlear membranes. Those affecting balance are called vestibulotoxic, because changes are found in the semicircular canals. The signs and symptoms of ototoxicity are tinnitus, vertigo, loss of balance and loss of hearing. Once these are present, significant damage to the auditory and/or vestibular apparatus has already occurred. It should be noted that tinnitus usually, but not always, precedes other symptoms and is, therefore, a cardinal sign of an early lesion. More sophisticated and more accurate measurements of toxicity are now available and will be discussed later.

Several classes of drugs may be ototoxic. We will consider three - antibiotics, diuretics and a miscellaneous class.

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Antibiotics

The antibiotics primarily guilty of producing severe ototoxic reactions are the aminoglycoside group. The important members of the group are streptomycin, dihydrostreptomycin, neomycin, kanamycin, gentamicin, tobramycin and amikacin. They are used for serious gram negative infections, and for some staphylococcal infections. They have the same basic formula, two amino sugars linked by glycoside bonds to streptamine (Fig. 1). These drugs are also extremely nephrotoxic and may produce renal tubular necrosis, especially in patients with underlying renal disease. It is not known whether the renal tubular effects are chemically identical to those on the ear. There is no valid information that explains why this particular chemical configuration is ototoxic, nor is it known which part of the molecule produces the effect. Streptomycin and dihydrostreptomycin are classic examples of ototoxic drugs. The former is vestibulotoxic in high doses. The latter causes profound deafness and, therefore, has largely been dropped from the therapeutic armamentarium. As a general rule the aminoglyosides are more likely to cause vestibular toxicity before hearing becomes affected.[2] The two exceptions to the rule are dihydrostreptomycin and neomycin. Neomycin is so cochleotoxic that it is rarely used parenterally. Even topical neomycin, used for burn therapy, has produced deafness, although there have been no reports of ototoxic reactions to topical therapy for minor skin infections.

Toxic reactions to aminoglycosides may occur at any time during the course of therapy. It is most discouraging to report cases of permanent deafness beginning as long as several weeks after the cessation of therapy. In other words, toxicity may be delayed. The levels of the drug in the blood, cerebrospinal fluid and in the

perilymph, which is one of the inner ear fluids, have been studied in experimental animals. It has been found that perilymph blood levels are the highest and that they peak later and fall later than the levels in blood or CSF. With repeated doses of medication the concentration of drug in perilymph may continue to rise despite the absence of drug activity in the blood during the interval between doses and may remain at a high level for several weeks. Renal function is the critical limiting factor in the development and maintenance of serum levels of drug. The aminoglycosides are almost totally excreted by the kidney, and this fact may be used to help determine and adjust safe dosages.

The histologic changes that have been identified in early cases of toxicity from kanamycin, gentamicin, tobramycin and amikacin are destruction of the hair cells of the semicircular canals. Later on the hair cells of the organ of Corti, the hearing organ, are destroyed. These histopathologic changes correspond to the changes in function of the ear.[3] Other antibiotics such as vancomycin, viomycin and capreomycin are ototoxic but are not widely used at this time.

Diuretics

Two potent diuretics, ethacrynic acid and furosemide, may cause deafness of either a transient or permanent type.[4] These drugs are classified as loop inhibitors - they block the reabsorption of sodium along the entire ascending limb of the loop of Henle. In the cochlea there is a structure called the stria vascularis which is thought to be the site of production and absorption of endolymph, one of the inner ear fluids. It is postulated that the effects of these two diuretics on the stria vascularis may be the same as on the loop of Henle, and definite inhibition of electrolyte transport resulting in changes in the sodium and potassium concentration of the endolymph has been shown. Hearing impairment has been associated with massive doses of these diuretics. By far the most potent is ethacrynic acid, and this drug is also responsible for most of the cochleotoxic effects. Ethacrynic acid also exhibits the characteristics of synergy, especially when used with gentamicin, and the combination of these two potent drugs should be avoided as no safe dosage levels can be relied upon.

Miscellaneous Drugs

Salicylates and quinines occupy a miscellaneous drug group. It is common knowledge that aspirin, even in usual therapeutic doses, may cause ringing in the ears as well as deafness of a temporary nature. Salicylate poisoning has produced permanent deafness, probably the result of a direct toxic effect on the ear. Quinine in small doses may cause tinnitus. In highly susceptible individuals even the small amount of quinine ingested in one or two average gin-and-tonics may produce symptoms. Chloroquine may cause permanent deafness. The site of the lesion is probably different than that affected by antibiotics or diuretics and may be a direct toxic effect similar to salicylates. In infants whose mothers have been treated with quinine during pregnancy, both congenital deafness and anomalies of the ear have been reported.

Diagnosis

The diagnosis of ototoxicity is a real dilemma - a "Catch 22" situation. Usually the problem is not identified until after the damage has been done, and the catch is that the damage may occur even weeks after the drug has been discontinued, probably due to slow clearance of drug from the inner ear fluids. Trying to identify a toxic reaction in a bedridden, seriously ill patient is difficult and yet this is precisely the type of patient in whom toxic drugs are most likely to be used. Tinnitus is a common complaint and it is often not associated with a toxic reaction of the cochlea.

The two most valuable early indicators of ototoxicity are the audiogram and the electronystagmogram or ENG. The ENG records for later comparison the responses obtained during caloric and positional stimulation of the ear. It measures the ocular nystagmus that is produced and is the only method presently available that records it. A pre-treatment audiogram and ENG provide a good baseline of auditory and vestibular function. Serial testing during the course of treatment and a post-treatment test will help to both identify early ototoxic reactions and to document the continued integrity of the auditory and vestibular organs, which may be important medicolegally. Toxicity will be demonstrated as either a neurosensory-type hearing loss or a paresis of the semicircular canals on one or both sides.

At the present time basic audiometric tests are widely available and should be obtained prior to treatment. The hearing loss produced by all three classes of ototoxic drugs usually begins in the high frequencies. The critical speech frequencies in the 500-2,000 Hertz range are not affected until the lesion becomes advanced, therefore, an early high frequency hearing loss may be an extremely important finding. A pre-treatment ENG should be considered if one of the more highly toxic drugs is contemplated, or if the course of therapy will be unusually long. Performing either examination on a seriously ill hospitalized patient is not very practical, accurate or reproducible, but again these are the patients who most often will develop drug-related problems. If hearing deteriorates, a decision will have to be made whether to decrease or discontinue the drug dose. At any rate, the patient must be informed of the potential adverse effects.

Prevention

Prevention is clearly the key; proper dosage schedules are the only type of control presently available and practical. Adhering strictly to the manufacturer's recommended dose schedules seems to be the best insurance against potential damage. Renal function tests, such as the BUN, creatinine and the creatinine clearance, may be correlated with the patient's body weight to adjust the total dose of drug or its frequency of administration. Nomograms have been prepared and may easily be followed. The main weakness in these methods is that the effects of decreased renal function on the concentration of a substance in blood is supposed to correlate with the concentration of another substance in perilymph, but there is limitation in the dimension of time. To be more accurate, bioassay and radioimmunoassay technics are available to measure the actual serum levels of drugs.[5] The therapeutic serum level is usually two to eight times the minimum inhibitory concentration (MIC) of the drugs, as determined by bacteriologic pharmacologic studies. By insuring that the serum levels are within the therapeutic range the chances of an ototoxic or nephrotoxic reaction are greatly reduced. Assays should be done whenever available, especially at the time of the peak serum level and at the valley level on the first day of therapy and then every subsequent few days. The peak serum level usually occurs 15-30 minutes after an intravenous injection or about one hour after an intramuscular injection. The valley level then will be that which occurs immediately prior to the next dose of medication.

Summary

Ototoxic reactions to potent antibiotics and diuretics are a real threat. The incidence may be as high as two per cent of patients treated. Reactions may occur several weeks after the termination of drug therapy. Synergism occurs between ethacrynic acid and the aminoglycosides, limiting the indications for combining these drugs Bioassay and radioimmunoassay of serum antibiotic levels are highly accurate. By maintaining a therapeutic serum level and by monitoring renal function to adjust the dosage of medication, maximum safety is obtained. Since some ototoxic reactions will be an inevitable consequence of urgently needed therapy, baseline audiometric and, less often, vestibulometric (ENG) examinations should be obtained whenever practical.●

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Brown spider bite*

Treatment with hydrocortisone

John E. Mara, MD, and Burton S. Myers, MD, Denver.

The brown recluse spider, Loxasceles reclusa, causes a significant medical problem in Colorado in the Autumn of each year. As the outdoor temperature falls, the spider frequently seeks refuge within heated buildings. This occurrence leads to an increase in the number of spider bites. Two such cases are reported here. One proceeded to cutaneous necrosis, and the other, recognized early, received steroid therapy immediately after diagnosis.

CASE REPORTS

Case 1: A 56-year-old male, while moving a cord of firewood into his house this past fall, experienced a small stinging sensation just above his hiking boot on the right ankle. He continued his project, and that evening noticed slight swelling and pain in the area. In the morning the ankle was markedly edematous and the skin tense with blue mottling. He was seen in the emergency room eighteen hours after envenomation, which it was determined was by L. reclusa, and was placed on hydrocortisone 100mg I.M. every eight hours for forty-eight hours. The lesion slowly progressed until a necrotic eschar, 4.0 by 5.0 cm., developed ten days later. This area was excised and subsequently skin grafted.

Case 2: A 52-year-old female, was bitten by a small brown spider on her right cheek while watching television. This spider was later identified as L. reclusa. Earlier in the day she had removed cobwebs from her porch. She was seen in the emergency room four hours after envenomation. Her right cheek was edematous with slight bluish discoloration. When examined under magnification (Fig. 1), this pale area contained two minute puncture wounds. The patient was immediately given 100mg of hydrocortisone intravenously, and ice packs were applied to her face. Sedation, elevation of the head, and 100 mg of hydrocortisone every eight hours were continued for the next twenty-four hours. Maximal edema and ecchymosis were present twenty-four hours after envenomation. The edema and discoloration resolved in ten days, leaving a small organised, firm hematoma in her upper cheek. This hematoma was slowly reabsorbed in six weeks.

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Pathophysiology

The brown recluse spider measures 3/8 inches in length and 3/16 inches in width. Color varies from deep mahogany brown to light fawn brown. The most distinguishing mark is a dark violin-shaped band on the anterior portion of the carapace which narrows to a thin line extending along the abdomen. The spider is therefore known as the "fiddle-back spider". Its webs are irregular and of medium size consisting of loose threads in all directions.

The venom of L. reclusa contains three significant compoments proved by electrophoretic analysis of microdissected venom glands.[1] Hylauronidase permits early flow of the dermonecrotic and esterase factors throughout the tissues, and allows spreading of the venom over a considerable area within hours of the bite. The dermonecrotic factor produces thrombosis and sludging in the small dermal vessels, presumably by activation of the clotting mechanism. This causes gangrene of the tissues by ischemia. The earliest change in the skin is edema in the corium with poly-morphonuclear leukocytes at the dermal-epidermal junction. Microthrombi begin to appear as the venom advances by diffusion through the tissues. In two weeks an eschar with sharp margins delineates the viable from necrotic tissue. The eschar will slough spontaneously in five to eight weeks if debridement and skin graftings are not performed in the interim.

A direct hemolytic effect produced by the venom against human erythrocytes has been shown in vitro.[2] Systemic effects in humans have been reported as consisting of fever, chills,

nausea, vomiting, and joint pain. Edema, jaundice, and convulsive disorders may occur in children.

Treatment

Various drugs have been prescribed to negate the effects of the venom. Benadryl, phentolamine and low molecular weight dextran were not effective in animal models. Antivenom was shown to inhibit completely the hyaluronidase effect when rabbit Gamma globulin was used in vitro. Usually the diagnosis is not made until at least twenty-four hours after envenomation when well-demarcated, necrotic areas are present, as illustrated by Case 1. Steroids have been used in the past from eight to twenty-four hours after venom injection. Dillaha et al found that in rabbits if steroids were given eight hours following injections, the systemic effects were aborted, but no effect on the tissue was noticed when steroids were used twenty-four hours after injection of the venom.[4]

Early institution of steroid therapy, as presented in Case 2, in our opinion will ameliorate the effects of the L. reclusa bite. The alternative treatment, again dependent on early recognition of the bite is total excision of the envenomated area and immediate skin grafting, a treatment similar to that prescribed for rattlesnake bites but not yet reported for spider bites. This technic would be applicable to areas such as the leg but not to the hand or face where significant tissue debridement is not permissible. If there has been early recognition of the envenomated area, local excision would appear to rid the body of the major portion of the venom before the hyaluronidase factor begins to work. The most effective overall measure in treating the brown spider bite would be the development of an L. reclusa anti-venom from human serum.

Fig. 1. L. reclusa bite on right cheek four hours following envenomation.

Summary

Two cases of envenomation by L. reclusa are presented. The cases differ in location of the bite and the time interim prior to therapy. Early recognition and institution of large doses of hydrocortisone help ameliorate the dermonecrotic effect of this venom. •

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Erythrocyte t activation*

Jennifer Bibbing, MT and Blaise E. Favara, MD, Denver

All erythrocytes possess a surface T receptor or antigen which is normally masked by a coat of siliac (N-acetylneuraminic) acid. An antibody (anti T) to the receptor is formed at about six months of age and can be demonstrated in fresh serum of most persons beyond that age. This antibody will not react with the T receptor unless the receptor is unmasked. Such reactions are referred to as polyagglutination since agglutination occurs with some but not all "normal" sera. The T receptor is unmasked and polyagglutination occurs in-vivo in association with certain bacteremias featuring organisms which produce the enzyme neuraminidase. This enzyme removes the erythrocyte sialic acid coat which covers the T receptor, thus freeing it to react with anti T present in serum.

Rarely spontaneous in-vivo T activation can occur in the absence of bacteremia. The phenomenon can also evolve in-vitro when red cell samples are contaminated by certain bacteria.

A case of a nine-month-old boy with pneumococcal meningitis, T activation and polyagglutination is reported here to illustrate the value of the minor crossmatch in blood-banking for such patients and to provide a basis for discussion of this phenomenon and the possible clinical implications.

CASE REPORT

A nine-month-old white boy was admitted to the Children's Hospital, Denver with a referral diagnosis of bacterial meningitis. He had been in good health until three days before admission when he developed fever and irritability and at which time physical examination and cerebrospinal fluid study demonstrated meningitis due to Streptococcus pneumoniae. Blood cultures were not taken, and the child was treated with ampicillin, 400 mg/kg/day intravenously for one day (9.52 kg for a total dose of 3.8 gm), and after the organism was identified, penicillin G, 200,000 units/kg/day intravenously for 11 days.

The hematocrit had fallen from 31.3 Vol.% on the day of admission to 23.8 Vol.% four days later. The anemia was microcytic-normochromic, but was not evaluated further. Many erythrocyte rouleaux were noted on examination of peripheral blood. A transfusion was given.

The patient was referred because of seizures for which he was treated with diazepam, phenobarbital, and paraldehyde, as needed.

The child's blood was typed as Group 0 Rh negative. The modified D typing was confirmed by saline D typing and the D test was also negative. The direct test was negative using broad spectrum antiglobulin reagent. No atypical antibodies were detected in saline at 22°C; albumin at 37°C or by indirect antiglobulin technic. When a unit of 0 negative blood was tested against the child's blood, there was 4 + incompatibility in the minor crossmatch on immediate spin at 22°C (donor's plasma with 5/ patient's erythrocytes suspended in saline). The major crossmatch was compatible in saline at 22°C; albumin at 37°C and by indirect antiglobulin technic. The patient's cells were further tested with four samples of serum from normal blood donors, all of which resulted in 4 + agglutination on immediate spin at 22°C.

T activation of the patient's erythrocytes was suspected in view of the diagnosis of infection. Further tests with cord serum and Arachis hypogea lectin were performed as shown in Table 1. The presence of T activation was thus confirmed.

The child was transfused with erythrocytes washed three times in saline in an automatic cell washing system. He had no ill effect from the transfusion, and the anemia was corrected successfully. He was discharged in good condition 17 days after admission. Fifteen days after transfusion the child's erythrocytes showed slight T activation and two months later there was no evidence of T activation or hematologic abnormality.

The patient's erythrocytes in suspension and when agglutinated in the presence of fresh serum were examined by scanning and transmission electron microscopy. No morphologically evident defects in the surface of erythrocytes were demonstrated by these technics. (Fig. 1)

Discussion

Polyagglutination is a phenomenon wherein erythrocytes are agglutinated by many but not all human serum specimens. The first description of polyagglutination was in 1925 by

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Hubner whose name is associated with its designation, the Hubner-Thomsen-Friedenreich phenomenon.[1] It was shown later that the phenomenon was the result of bacterial effects in vitro or in vivo when those organisms produced the enzyme neuraminidase (sialidase). This enzyme is believed to be capable of exposing a hidden antigen-like receptor, the T receptor, normally found in the erythrocyte or the cell membrane. All normal erythrocytes carry the T receptor and virtually all normal adult serum contains an antibody to this receptor in varying amounts.[2] It has recently been shown that the T antigen or receptor also occurs in soluble form in normal saliva and in neuraminidase-treated serum and saliva.[3] The antibody anti-T is formed at approximately six months of age and its development coincides with colonization of the gut by coliform organisms.[4] Serum from young infants and cord samples contain little or no anti-T.

The phenomenon of polyagglutination depends upon the unmasking of surface T receptor brought about through the action of an enzyme which removes the N-acetylneuraminic acid coat from the surface of the erythrocyte. Erythrocytes so induced to T activation have reduced amounts of N-acetylneuraminic acid on their surface. The degree of T activation of a cell depends on the amount of surface coat removed. Titers of anti T also vary in different serum samples so that the intensity of polyagglutionation is dependent on these two factors.

In the blood bank point, difficulty may be encountered in grouping of cells that have been T activated in vivo since reagent antisera may contain anti-T. When such a problem is encountered, methods for absorbing anti-T from antisera using neuraminidase treated 0 negative cells must be employed. Anti-T may not be demonstrated in the serum of patients whose cells exhibit T activation, since under certain conditions the combination of anti-T with T antigen in serum or on the erythrocyte is sufficient to deplete the antibody from serum.[3]

Specific confirmatory testing for T activation depends on the use of a lectin derived from an extract of peanuts (Arachis hypogea).[5] This lectin has specific anti-T activity and can be used to establish the presence of T activation in a given red cell population. The profile used for confirming T activation is shown in Table 1. It must be emphasized that T activation may go undetected unless the minor crossmatch is carried out.

TABLE 1

T Activation - Confirmatory Testing

	Agglutination Results		
	Cord Serum	Normal AB Serum	Lectin*
Normal O Cells	0	0	0
Neuraminidase-treated Normal cells	0	4	4
Patient's cells	0	4	4

*Lectin-Arachis hypogea

The transfusion of patients with T activated erythrocytes poses a potentially dangerous situation, at least one patient having been reported to have suffered a severe transfusion reaction following infusions of whole blood. Since the reaction is one resulting from transfusion of antibody to T receptor., the chances of reaction can be minimized with the use of saline-washed erythrocytes with essentially no plasma protein component in the transfused material.

Transfusion of leukocyte and platelet concentrates, products which are both suspended in plasma, also carries the risk of inducing reactions involving T activation. Since leukocyte transfusions are generally given to patients with infections, and such patients could possibly have T activation, the plasma in such infusions may result in polyagglutination and reaction. Again, a minor crossmatch is essential to detect such a potential adverse effect of transfusion.

The role of T activation in induction of anemia in patients with infection is not entirely clear, however, one could speculate that polyagglutination attendant to T activation of sepsis may play a role in the induction of anemia which is so common in these conditions. The process would be somewhat self-limited by the temporary nature of exposure to neuraminidase produced by microbes and by the consumption of anti-T during the process of polyagglutination.

The fate of the antigen-antibody complexes formed by the interaction of anti T antibody with serum T antigen is uncertain but renal trapping and renal dysfunction is possible.

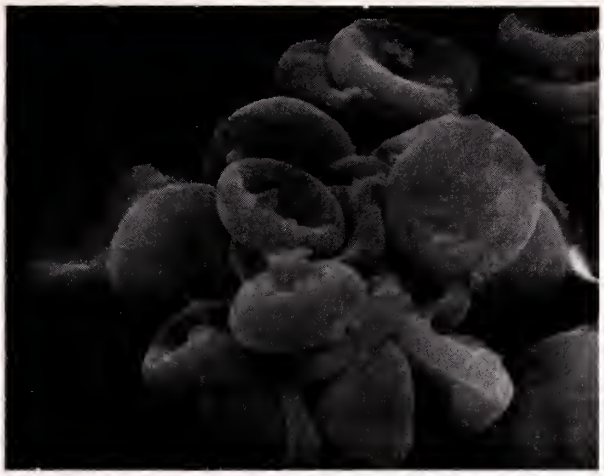

Fig. 1. Scanning electron microscopy 4000x photograph shows clusters of normal appearing erythrocytes agglutinated after T activation with normal serum.

Summary

A case of a nine-month-old boy with bacterial meninigitis who had T activation of his erythrocytes is presented. The detection and confirmation of T activation is described, and the relationship of this phenomenon to transfusion reactions and perhaps anemia associated with infection is suggested. The essential role of the minor crossmatch for detecting such phenomena is emphasized.●

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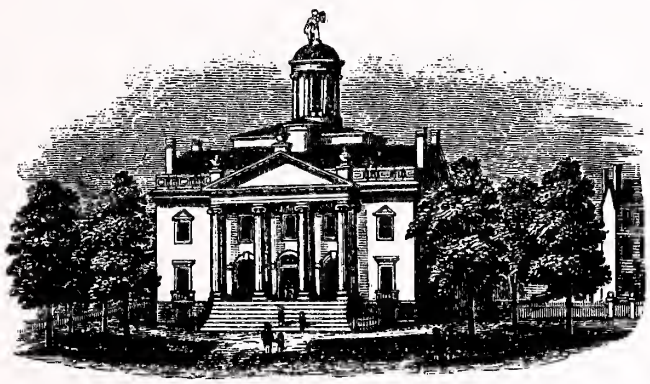

East View of the Capitol at Albany.

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Please include beneath the title or at the top of the first page a brief paragraph stating what the article is designed to show and, therefore, why it should be read. This introductory feature is, in editorial parlance, called the "blurb"; it is printed in bold type.

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4. Acceptance or rejection. The state editor will (1) tentatively accept the article, (2) return it to the author with suggestions for revision, or (3) reject it. He will edit and forward approved copy to the publication office in Denver for final editing and scheduling.

5. Order of publication. Ordinarily, articles will be published in the order received. Those whose value is seasonal, whose value would be lost if publication is delayed, or which reflect priority of original research, are given special consideration. State presidential addresses, programs, state society minutes, and timely organization announcements are given priority.

6. Length of articles. Scientific papers should be "boiled down" not to exceed ten pages of double-spaced typewritten standard 8½ x 11 inch sheets. Shorter papers are more acceptable to editors and readers, and earlier publication is probable. Every journal is in competition for the reader's time, and condensation is the order of the day. For this reason, and because of financial and space limitations, we request that copy be condensed to the greatest extent consistent with conveying the message.

7. Illustrations. A limited number of illustrations or "cuts," usually up to six, will ordinarily be accepted within our own publication budget if the editor believes they enhance the value of the article. Clear photos, simple diagrams or line drawings in black on white, printing rather than writing, reproduce well. Cuts should be mounted separately, and the paper or cardboard mounts should be the same size as that upon which the article is typed. Each should have its caption below: Fig. 1, Fig. 2, etc., with a short descriptive sentence. Authors will be billed our cost for excess above six cuts.

8. Tables. Tables should be simple, presenting only brief relevant data, amply spaced. They should be placed at the back of the manuscript on separate sheets. Each should have its number and title above: Table 1, Title; Table 2, Title; etc. Long, large, or complicated tables ordinarily are not acceptable.

9. Case reports. Case reports are popular with our readers. A brief introductory statement concerning the condition or disease, tells why the case is presented. The report follows with a separate heading, CASE REPORT or CASE 1, CASE 2, etc. Include only relevant, positive laboratory and other data. Names, initials, and numbers are unimportant; age, sex, and sometimes occupation are significant. Minimize dates; then make it month, date, year—as February 9, 1969; not 2/9/69. After the case presentation, review or sum it up under a heading such as Discussion, Comment, Summary or Conclusion.

10. Footnotes. Footnotes must be brief. One at the bottom of the first page should state the Society and date of presentation, and institutional origin, if any. Special titles or position of the author, acknowledgements, etc., will be added according to simplicity, editorial policy and discretion.

11. References. Reference lists rarely add to the value of an article for the majority of readers. We will make every effort to publish short reference lists, but long ones will be deleted. Incidentally, the proper heading is "References," not "Bibliography." The latter implies everything in the literature upon the subject. Make them uniform in style and brief, as: Jones, W. K.: Pyelonephritis in Infancy. JAMA 141:75. 1964.

12. Editorials. We would like to have more submitted by our State editors and the membership at large. Readers must get tired of thunder from publication headquarters in Denver; frankly, we do too! Speak out, and we'll sign your name, initials, or respect your request for anonymity, as you choose. If you don't like something we do or say, please let us in on it; we could even stand to hear about it if something happens to please you. A "Letter to the Editor" may help you get something off your mind. We're asking for it and we have a place to put it—in the Journal, that is!

13. Proofs. Galley proofs are submitted to the author, and prompt return is essential. Authors should correct typographical, grammatical, or rhetorical errors, but do not reinsert or rewrite sentences, paragraphs, tables, etc., which an editor may have deleted, condensed, or paraphrased. When an article reaches the galley-proof stage, the author may NOT re-edit his article; that is the privilege of the Journal's editors.

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Neck masses:
*a diagnostic approach**

Arlen D. Meyers, MD, Denver

No mass in the neck should be biopsied until the possibility of a distant primary disease, particularly in the aerodigestive system, has been excluded.

Evaluating and treating a patient with a mass in the neck is a common problem. The urge to biopsy the mass at once must be resisted in favor of a systematic, logical approach to the patient's problem. Several questions should be answered before any such mass is biopsied. For example, given the patient's history and physical examination, is it likely that the mass is malignant? If metastatic, where are the most likely sites of the primary lesion? What diagnostic procedures should be performed prior to biopsy to help delineate the lesion? Will initial biopsy interfere in any way with subsequent therapy?

The following is a format for diagnosis of a mass in the neck. Utilizing simple technics of palpation, inspection, and routine indirect endoscopic methods, all physicians caring for patients with head and neck masses should be able to formulate an initial diagnostic protocol and make the appropriate referral if need be. Only until the technics of examining the head and neck are mastered can the physician intelligently manage a patient with a mass in the neck. Indeed, the large majority of patients with neck masses will require radiologic examination, endoscopic procedures, and random biopsies of the pharynx prior to excision of the mass.

* From the Division of Otolaryngology, Denver General Hospital, and the Department of Otolaryngology, University of Colorado Medical Center, Denver, Colorado.

Why Not Biopsy?

Several authors have pointed out that in certain patients most persistent neck masses are malignant, most of these malignant tumors are metastatic, and the primary site is usually in the head and neck.[1] If the mass is malignant initial biopsy may interfere with therapy in several important ways.

Often times, following a biopsy of a malignant mass in the neck, subsequent evaluation and therapy requires diagnostic procedures not available to the primary physician, thus delaying therapy of the primary disease. In addition, dissemination of tumor cells at the time of the biopsy is theoretically undesirable and could contribute to a higher incidence of persistence. Perhaps the scarring from the biopsy will prevent a clean dissection, should it be necessary, or more importantly, the previous biopsy incision may complicate the planning of a subsequent neck dissection incision particularly by jeopardizing blood supply to flaps. Should radiation therapy be utilized, the tumor in the area of scarring is less likely to be sterilized because of surrounding anoxia of tumor cells. Indeed should the disease of the neck originate from a primary process which is radiocurable, such as carcinoma of the nasopharynx, then surgery is not only ill advised but meddlesome.

Is Mass Benign or Malignant?

Without histopathologic confirmation, of course, no one can say for sure whether a mass is benign or malignant. There are several factors in the history, however, which can help in differentiating between benign and malignant processes. Certainly a history of alcohol and/or tobacco abuse makes an individual suspect of harboring a malignant tumor. Although men

more commonly have head and neck cancer than women, there appears to be a tendency toward equalization in recent years. The age of a patient with a neck mass is important. Children have a different incidence of malignant neck tumors than adults, the types of tumors are different, and the differential diagnosis varies.[2] By and large, most malignant tumors of the neck in children are of the lymphoreticular type, whereas these of an adult are usually squamous cell carcinomas or adenocarcinomas.

Prior history of irradiation to the head or neck contributes to subsequent development of malignant disease, particularly of the thyroid, and this possibility should be explored. In addition, a history of change in the size or characteristics of the mass, surrounding erythema, or associated inflammatory signs will help differentiate between inflammatory and neoplastic disease. Prior trauma or surgery to the neck, systemic disease, occupational history, and other associated symptoms such as a change in voice or hearing likewise aids in diagnosis. A mass present for several years without change in size makes a malignant process unlikely, but still a possibility.

Importance of Location

The location of the mass in the neck is one of the most imporatant factors in formulating an approach to diagnosis. Masses in the midline, particularly in children, tend to be benign. Although thyroid tumors do present in the midline, such benign lesions as cysts, dermoids, pyramidal lobe of the thyroid, and other things are more common. (See Fig. 1). If the mass is in the lateral part of the neck, then its precise location is important. The lymphatics of the head and neck essentially form a circle of nodes, like a collar, around the neck. From this primary chain, three secondary chains descend in the lateral part of the neck in a triangular fashion-the internal jugular chain, the accessory chain, and the supraclavicular chain. Characteristic tumors of the head and neck will metastasize to certain lymphatic structures in a fairly regular fashion. Thus, a node in the upper jugular node would make a lesion in the pharynx, base of tongue, or tonsil suspect. A supraclavicular mass on the other hand, usually is associated with disease below the clavicle, most characteristically breast, lung, or gastrointestinal system.

TABLE 1

Differential Diagnosis of a Lump in the Neck

1. Cancer
   - A. Primary
   - B. Metastatic
     1. From primary of the head and neck (60-70%)
     2. From primary other than head and neck tumor (20%)
     3. No primary found (5-10%)

2. Not Cancer
   - A. Benign tumor
   - B. Cyst
     1. Bronchogenic
     2. Parathyroid
     3. Thymic
     4. Thyroglossal
     5. Dermoid
     6. Branchial cleft
     7. Cystic hygroma
     8. Sebaceous
   - C. Developmental anomaly
     1. First arch
        - a. Type I
        - b. Type II
     2. Second arch
   - D. Ectopic tissue
   - E. Laryngocoele
   - F. Infection or infestations
   - G. Vascular lesions
   - H. Variant or normal structure
   - I. Other

What Else is Important?

If the mass in the neck is malignant there are several features peculiar to the mass that will affect the prognosis. Among these, the size of the mass, whether it is fixed to any adjacent structures, and whether there are multiple or bilateral masses are most important. If a mass in the neck is suspected of being malignant, then the disease should be clearly documented and staged according to some standard format. One such as authored by the American Joint Committee is offered in Fig. 2.

Several diseases manifest themselves in characteristic ways. A laryngocole for example, a cystic out-pouching usually arising from the saccule of the laryngeal ventricle, is an air-containing sac and is relatively compressible; bowel sounds may sometimes be heard over the mass after the patient swallows. Vascular lesions of the neck on the other hand, such as glomus tumors, are sometimes associated with a bruit or thrill. A cranial nerve palsy associated with a lump in the neck is a prognostic sign that should raise a red flag in the mind to the physician. A unilateral vocal cord paralysis, facial paralysis, or a shoulder drop, should it be recognized, requires intensive investigation. A unilateral serous otitis media or an isolated lateral rectus palsy may be the first representing sign of a nasopharyngeal tumor.

TABLE II

Neck Node Classification

NO: No clinically positive node.
N1: Single clinically positive homolateral node less than 3 cm in diameter.
N2: Single clinically positive homolateral node 3 cm to 6 cm in diameter or multiple clinically positive homolateral nodes, none over 6 cm in diameter.
N2a: Single clinically positive homolateral node 3 cm to 6 cm in diameter.
N2b: Multiple clinically positive homolateral nodes, none over 6 cm in diameter.
N3: Massive homolateral node(s), bilateral nodes, or contralateral node(s).
N3a: Clinically positive homolateral node(s), one over 6 cm in diameter.
N3b: Bilateral clinically positive nodes (in this situation each side of the neck should be staged separately; that is, N3b -- right N2a, left N1).
N3c: Contralateral clinically positive node(s) only.

It is clear from the above that a complete and thorough evaluation of all of the mucous membranes and the skin of the head and neck should be visualized, palpated, and in some instances listened to, as part of the initial work-up of the patient with a mass in the neck, regardless of the patients age. This includes a careflul palpation of the scalp, visualization of the ear canals and tympanic membranes, careful examination of the nasopharynx, oropharynx, hypopharynx and larynx. Any abnormal eye symptoms should be pursued and the fundi adequately visualized.

Suppose Head or Neck Primary Lesion Presents?

It's obvious that every patient with a lump in the neck will require different diagnostic efforts. One must consider all the possibilities and, utilizing knowledge and clinical judgment, utilize those diagnostic efforts which will be most rewarding. In general, an adult with a persistent mass in the neck should have a chest x-ray, soft tissue neck, sinus x-rays, barium swallow, upper GI, and thyroid scan performed proior to biopsy of the lesion. Such studies as laryngeal tomography or contrast studies, sialograms, salivary scans, or arteriography may sometime by indicated depending on the clinical situation. In addition, proper evaluation of the bone marrow, thyroid homrmone levels, and other chemistries may reveal valuable data.

If General Work-up is Normal

In most instances, the next step is endoscopy. Fifty per cent of carcinomas of the nasopharynx present with a neck mass, as do twenty-five per cent of patients with carcinoma of the pharynx.[3] Despite a normal radiographic appearance of the aerodigestive system, small neoplasms, either too small to be detected by x-rays or submucosal in nature, may be present. It is therefore essential that the patient have direct laryngoscopy, esophagoscopy, bronchoscopy (preferrably with a fibroptic bronchoscope), and careful examination of high risk anatomic areas while anesthetized. Should no obvious source of tumor be found, random biopsies of the nasopharynx, base of tongue, tonsil, and pyriform sinus should be performed, and carefully labeled. When taking a biopsy of the nasopharynx the mucosal area should be removed first, and a repeat biopsy then performed in the same region to insure obtaining a submucosal sample. Some advocate frozen section interpretation of the biopsy obtained, and this should be discouraged. In all fairness to the patient and the pathologist, the permanent section should be carefully scrutinized and a final diagnosis obtained. Only then can the physician intelligently present the treatment alternatives to the patient and allow him to partake in the theraputic decision.

Should Endoscopy be Negative

Only after complete history and physical examination, adequate laboratory and radiographic studies, and careful endoscopic scrutiny, shouid the neck mass be biopsied. The decision as to whether a needle biopsy or an incisional or excisional biopsy should be performed is to a large extent dependent on expertise and experience of the surgeon and pathologist. Although the morbidity of needle biopsy is minimal, the disadvantages are several in an institution unpracticed in examination of needle biopsy specimens.

In general, therefore, an excisional or incisional biopsy is performed. Some believe that, should the biopsy prove to be squamous cell carcinoma, the immediate neck dissection should be performed and this eventuality should be explained to the patient preoperatively.[4-6] Others believe radiation therapy is the treatment of choice.[7] It can be said, however, given the available data, no conclusive study exists which appears to answer the question as

to which is the best treatment for an "occult" malignant node in the neck. Until such factors as the natural history of the disease, adequate follow up, the stage of nodal disease, the presence or absence of other metastastes, and other general patient parameters, are assessed, no meaningful conclusion can be drawn between present comparative studies.

Summary

Most authors have found that approximately five to eight per cent of patients with a metastic lymph node in the neck will have a primary source which is undetectable.[8,9] In most cases the primary source will be found if properly sought. Frequent followup at appropriate intervals is mandatory once a malignant mass in the neck is diagnosed. With repeat examination and endoscopy, if need be, the etiology will be found in an overwhelming majority of cases. Those caring for individuals with neck masses are behooved to adhere rigidly to the following principles:

a. Complete and thorough evaluation of the aero digestive system, either directly or radiographically when appropriate.

b. Awareness of the serious nature of most persistent masses in the neck.

c. Consultation with individuals familiar with modern treatment modalities of cancer of the head and neck.

d. Excision of a neck mass only as a final diagnostic maneuver. •

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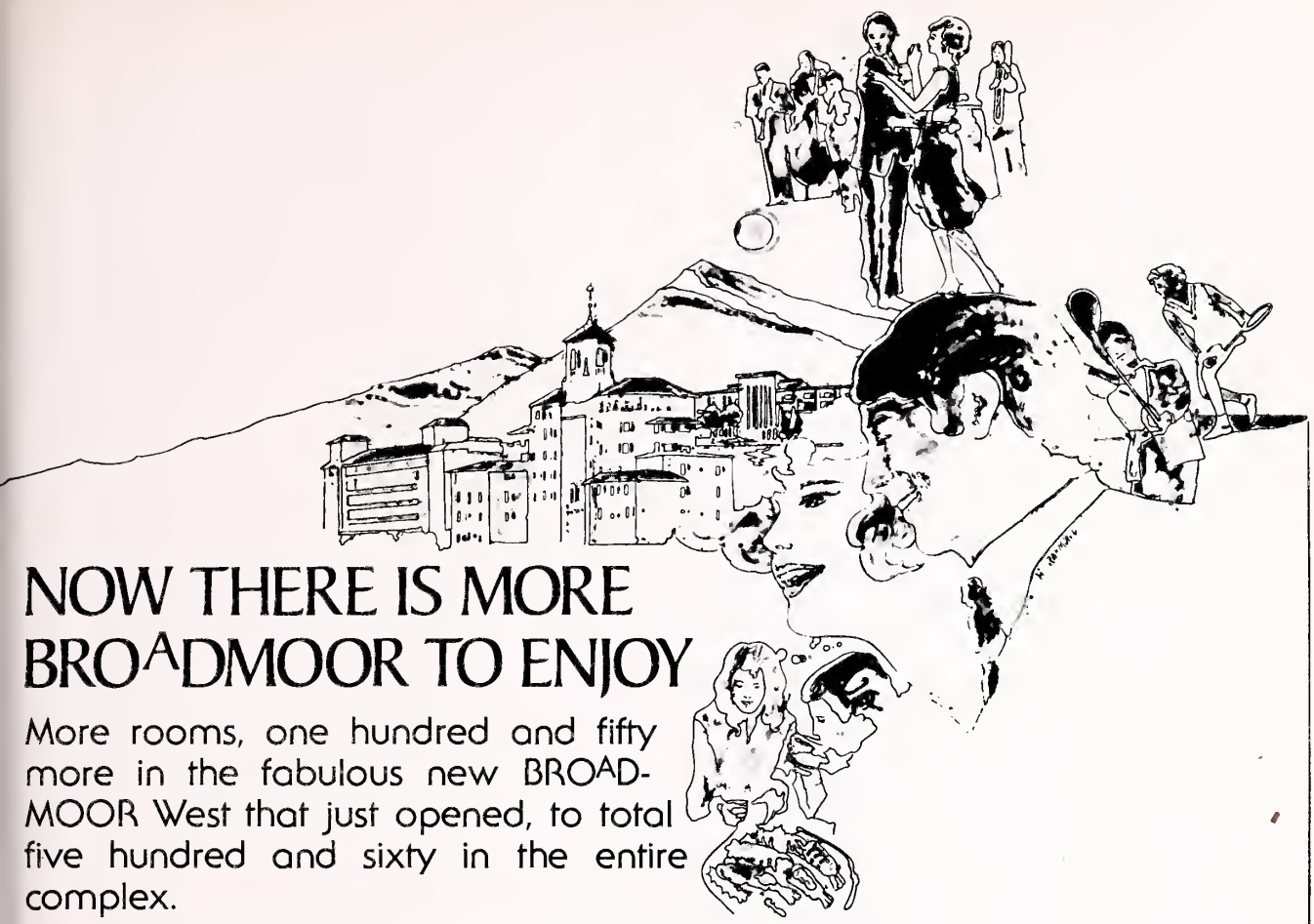

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Vitamin D	75	75	75
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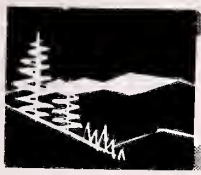

COLORADO

Dr. Pierce Named President of Pathologists

G. Barry Pierce, MD, professor at the University of Colorado School of Medicine and chairman of the Department of Pathology, has taken office as president of the American Association of Pathologists. The organization, created in 1976 through the merger of the American Association of Pathologists and Bacteriologists and the American Society for Experimental Pathology, requires evidence of creditable research in pathology.

Dr. Ritsick Joins Spalding Staff

Joseph A. Ritsick, MD, Board Certified Physiatrist, has come from the Institute of Rehabilitation Medicine at Good Samaritan Hospital, Phoenix, Arizona, where he was Staff Physiatrist, to serve in the practice of Physical Medicine and rehabilitation at Spalding Rehabilitation Center.

Dr. Ritsick received his MD from Hahnemann Medical College and Hospital, Philadelphia, Pennsylvania; completed his internship at Allentown Hospital, Allentown, Pennsylvania, and fulfilled residencies in Physical Medicine and Rehabilitation at Thomas Jefferson Hospital, Philadelphia, and in Internal Medicine at Allentown Hospital.

From 1974 to 1976 Dr. Ritsick was on active duty with the Army Medical Corps, serving as Assistant Chief of the Physical Medicine and Rehabilitation Service at Fitzsimons Army Medical Center.

Denver Hope Director In U.S. for Meet

Burris Duncan, MD, of Denver, Program Director for Project HOPE'S medical education program in Natal, Brazil, recently attended the Third Annual Project HOPE Directors Conference in Washington, D.C. He formerly was a pediatrician at the University of Colorado Medical Center, and received his MD at the University of Kansas Medical Center. He had served with Project HOPE in Maceio, Brazil in 1973.

Glenwood Springs Doctor Attends Eyes in Southern Africa

Roy W. Day, MD, Glenwood Springs, Colorado, joined the Peace Corps in 1975 after 36 years of medical practice, and has been serving in the Kingdom of Lesotho, a nation of one million entirely surrounded by the Republic of South Africa. Dr. Day served at the Maluti Adventist Hospital which is fifty miles NW of Maseru, capital city. Since coming to Lesotho in 1975 Dr. Day has performed about 400 eye operations, mostly cataracts. Two days a week he travels 22 miles on to a clinic and another 50 miles distant. Dr. Day's term was concluded in July 1977.

Dr. Curran Named Orthopaedic President-Elect

Thomas E. Curran, MD, Aurora physician, was named Vice President/President Elect of the Mid-Central States Orthopaedic Society, Inc. during the 1977 annual meeting at Cedar Rapids, Iowa in May. Dr. Curran will succeed President Worth M. Gross of Tulsa, Oklahoma.

Rhodes Edits Book

Buck A. Rhodes, PhD, director of radiopharmacy in the College of Pharmacy, has edited a 544-page volume, "Quality Control in Nuclear Medicine," which includes the works of 62 authorities.

Nelson Addresses Society

William R. Nelson, MD, of Denver, outgoing president of the Society of Surgical Oncology, delivered his Presidential Address during the Annual meeting of the Society, held in conjunction with the Society of Head and Neck Surgeons, at Hilton Head Island, South Carolina in May.

MEDICAL SCHOOL NOTES

University of Colorado Medical Center

Holmes Retirement Banquet Reset

The dinner to honor retiring UCMC professor Joseph H. Holmes, MD, originally set for May 25, but postponed because of Dr. Holmes' illness, has now been rescheduled for Friday, October 28, at the Denver Hilton. Dr. Holmes retired on July 1, 1977 after 42 years service. In his honor, the Joseph H. Holmes Visiting Professorship in Innovative Medicine is being established through contributions. Such funds are being accepted by the Joseph H. Holmes Fund, Box C-277, University of Colorado Medical Center, 4200 East 9th Avenue, Denver, Colorado 80262.

Edwards Elected President

W. Sterling Edwards, MD, chairman of the surgery department, has been elected president of the North American chapter of the International Cardiovascular Society, composed of 500 cardiovascular surgeons in the U.S. and Canada.

Satellite Transmittal of Biomedical Information Planned

One of four institutional sites in a federally funded experiment to transmit medical programs via Communications Technology Satellite is the University of Colorado Medical Center. A joint experimental program of the Canadian Department of Communications and the U.S. National Aeronautics and Space Administration to explore applications of advanced satellite technology to communications, the program makes use of ground stations at the National Library of Medicine, Bethesda, Maryland; the University of Kentucky, Lexington; the University of Washington, Seattle.

Five projects will be developed by the University of Colorado Medical Center during the next year. These include dental education, nursing child assessment satellite training experiment, dissemination of biomedical research information to health care practitioners and the public, an Association of American Medical Colleges experiment, and the American Diatetic Association experiment.

Pharmacology Head Named

Alan S. Nies, MD, professor of medicine and Pharmacology at Vanderbilt University School of Medicine, Nashville, Tennessee, has been named to head the Division of Clinical Pharmacology at the University of Colorado School of Medicine. Dr. Nies, a diplomate of the American Board of Internal Medicine, received his BS from Stanford University and an MD from Harvard Medical School, and has had a 2-year fellowship at the Division of Clinical Pharmacology and Cardiovascular Research Institute at the University of California, San Francisco. He was later chief of the Clinical Pharmacology Section of the Medicinal Chemistry Division at Walter Reed Army Institute of Research in Washington, D.C.

The Division, within the Department of Medicine, is involved with the effects of drugs on humans, and operates a drug assay laboratory to measure concentrations of drugs in patients to determine proper treatment.

University of New Mexico Medical School

New Mexico

Dr. Napolitano to Serve as Director

Leonard M. Napolitano, MD, has been named director of the University of New Mexico Medical Center, the former UNM Health Sciences, Dr. Napolitano has served as interim Vice President for Health Sciences at UNM since March 1976. Previously he was dean of the UNM School of Medicine.

Cauthorne Named Chief

Tom Cauthorne, MD, former chief of medical service at the Veterans Administration Hospital in Cheyenne, Wyoming, has been named chief of staff at the Veterans Hospital in Albuquerque, New Mexico.

Saiki Honored

John H. Saiki, MD, Associate Professor of Medicine at the University of New Mexico Medical Center, and Assistant Scientific Editor of the *Rocky Mountain Medical Journal* for New Mexico, has received the Khatali award from the UNM Medical Center for outstanding teaching.

Kellner Speaks

Robert Kellner, MD, chief of the Veterans Administration hospital psychiatry service and professor of psychiatry at the UNM Medical Center, served as a panel member in "Treatments of Personality Disorders" at the annual meeting of the American Psychiatric Association held in May in Toronto.

UNM Faculty Presents Panel

A panel on forensic medicine was presented during a Science Writer's Forum in New York City by James T. Weston, MD, chief medical investigator for New Mexico, and professor of pathology; Homer Campbell, DDS, clinical associate in pathology, and Byron G. Brogdon, MD, chairman of the radiology department.

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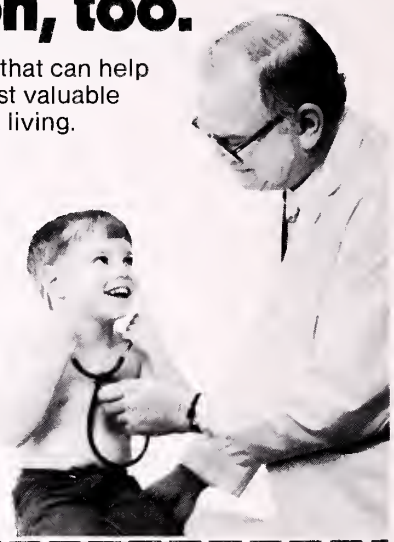

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☐ We do not have videocassette playback equipment

3rd COLORADO RURAL HEALTH CONFERENCE

October 28 & 29, 1977

Lion Square
Lodge and Conference Center
Vail, Colorado

PROGRAM

THURSDAY, OCTOBER 27, 1977

8:00 - 9:00 p.m. Registration for Conference - Meeting Room Lobby

FRIDAY, OCTOBER 28, 1977

8:00 - 9:00 a.m. Registration and Continental Breakfast - Meeting Room Lobby
9:00 a.m. Welcome and Orientation - Don Ervin, M.D., Conference Committee Chairman
10:00 a.m. Concurrent Presentations

A. Role of the University Medical Center in Rural Health. Past, present and proposed activities of the University of Colorado Medical Center relating to rural health problems will be discussed and considered in light of experiences in other states.
Speakers: John W. Cowee, Ph. D., Chancellor of the University of Colorado Medical Center
Amos P. Bretrude, M.D., Rural Family Physician and Clinical Associate Professor, WAMI Program, University of Washington Medical School

B. Urban Planning for Rural Needs. This session will address the issue of health care planning and who should do what. Many rural people feel that too much urban planning is done on behalf of rural folks. Many urban people feel that local planning produces fragmented and low quality services. There is a large "gray" area which is in dispute. The participants in this session will have an opportunity to hear both sides of this issue and question the presenters.
Speakers: Lee Wolsey, Executive Director, Northwest Colorado Council of Governments - Frisco
James McShane, Dr. H., M. P. H., Director Emergency Medical Services Division, Colorado Department of Health - Denver
Moderator: Mary Jo Jacobs, M.D., Colorado Medical Society Rural Health Committee - Glenwood Springs

C. Self-Care: Changing Responsibilities for Health. A growing nation-wide movement is challenging the individual to take more responsibility both for his own health and for the health needs of his community. Speakers will review the holistic health movement and focus in on the issues it raises about the consumer/provider relationship, use of non-medical community resources, and the problems of applying this idea of self-responsibility in the rural setting.
Speakers: Leland R. Kaiser, M.P.H., Ph.D., Acting Director, Division of Health Administration, Department of Preventive Medicine and Comprehensive Health Care, University of Colorado Medical Center
Merie Cunningham, M.D., M.P.H., Medical Director, Salud Clinic - Fort Lupton
David S. Meyer, Executive Director, Western Colorado Health Systems Agency

12:00 Noon Luncheon
1:30 p.m. Concurrent Workshops
5:00 p.m. Cash Bar
5:30 p.m. Deadline for submitting resolutions
8:00 p.m. Share Work and Programs (SWAP)

Time for individuals and organizations to share program innovations and information. Opportunity for small groups to discuss issues of mutual concern. Details concerning logistics and content areas will be announced Friday morning and updated Friday at lunch. For more information and scheduling, call Virginia Bell, Colorado Medical Society, Telephone: 534-8580.

SATURDAY, OCTOBER 29, 1977

7:00 - 8:30 a.m. Buffet Breakfast
9:00 a.m. Concurrent Workshops
12:30 p.m. Luncheon
1:30 p.m. Discussion of Resolutions - Conference Evaluation - Summation - Adjournment

New location - - new approaches - - providing a continuing forum for everyone concerned with rural health in Colorado. Focus will be on the following subjects:

* Clarifying the realities of the current situation
* Seeking consensus on health action recommendations
*Providing training and information in essential areas
*Facilitating information exchange

WORKSHOPS AND PRESENTATIONS

ASSESSING COMMUNITY NEEDS FOR HEALTH SERVICES
FINANCING COMMUNITY HEALTH SERVICES
IDENTIFYING AND USING EXISTING HEALTH RESOURCES
IMPACTING THE POLITICAL SYSTEM
COMMUNITY ORGANIZATION
RECRUITING AND RETAINING HEALTH PERSONNEL
RAPID GROWTH AND THE QUALITY OF LIFE IN RURAL COMMUNITIES
MENTAL HEALTH SERVICES IN RURAL AREAS
RESOLVING COMMUNITY CONFLICTS
SCHOOL HEALTH IN RURAL AREAS
IMPACT OF COST CONTAINMENT PROGRAMS
SELF CARE: APPLYING IT

OFFICIAL SPONSORS

Those who thus far have joined the COLORADO MEDICAL SOCIETY as co-sponsors are the following:

COLORADO ACADEMY OF FAMILY PHYSICIANS
ST. ANTHONY HOSPITAL SYSTEMS
RURAL HEALTH CARE ASSOCIATION
MENTAL HEALTH ASSOCIATION OF COLORADO
CENTRAL NORTHEAST HEALTH SYSTEMS AGENCY
COLORADO OSTEOPATHIC ASSOCIATION
COLORADO HOSPITAL ASSOCIATION
COLORADO REGIONAL CANCER CENTER, INC.
RICHARD D. LAMM, Governor, State of Colorado
.. Department of Health
. . Department of Institutions, Division of Mental Health
UNIVERSITY OF COLORADO MEDICAL CENTER
. . School of Dentistry
. . School of Medicine
- Department of Family Medicine
- Department of Preventive Medicine
. . School of Nursing
THE ACADEMY OF CHILD HEALTH ASSOCIATES
SOUTHEASTERN COLORADO HEALTH SYSTEMS AGENCY
COLORADO DENTAL ASSOCIATION
COLORADO OPTOMETRIC ASSOCIATION
BLUE CROSS/BLUE SHIELD OF COLORADO

Don L. Ervin, M. D., Chairman, Conference Committee

For further information and registration, write:

Mrs. Virginia Bell
Colorado Medical Society
1601 East 19th Avenue
Denver, Colorado 80218

$35 per person registration fee ($20 for full-time students and Senior Citizens) includes two breakfasts and two luncheons. Make checks payable to Colorado Medical Society. Room reservation should be made directly with Lion Square, 655 W. Lionshead Place, Vail, Colorado 81657.

OBITUARIES

Doctor Kenneth C. Sawyer died at his home in Denver on Friday, July 29, 1977. He was 72 years of age, born in Windsor, Colorado October 1st, 1904.

A graduate of the University of Colorado School of Medicine, he served his profession, community, and country unselfishly nearly half a century. He was the Colorado delegate to the A.M.A. from 1956 to 1969, having served as alternate for the preceeding seven years.

In 1970 he was elected to the A.M.A. Board of Trustees where he served until June 1976. He was the second Coloradan to be so honored. While a trustee, he served on or chaired numerous committees.

Throughout his active years with the A.M.A. he continued untiringly to support, guide, and advise his local and state medical societies. In addition, he served countless professional, community, and civic organizations while carrying on one of the most active surgical practices in the area. He authored more than 125 scientific articles in medical journals.

To list all the professional and civic organizations to which he belonged would be quite redundant, however, to mention a few: he was president of the Western Surgical Association in 1970, and of the Southwestern Surgical Congress in 1958, he was a Fellow of the American College of Surgeons and a Fellow of the American Medical Writers Association, he was one of the founders and past president of the Denver Academy of Surgery, he became a diplomate of the American Board of Surgery in 1940.

He was quite active in the American Cancer Society and in the Colorado division of that organization. He served on the Denver County Board of Health and Hospitals, being its chairman in 1965. He was a Mason, a member of the Denver Athletic Club and the Denver Country Club, and was quite active in the University of Colorado Alumni Association, having been honored repeatedly with awards from various branches of that organization. He received a Certificate of Service from the Colorado Medical Society in 1951 and again in 1976.

Doctor Sawyer had an active interest in teaching, having supported the teaching programs at the Presbyterian Medical Center throughout his career. He was Emeritus Chairman of the Surgical Service at that institution as well as Emeritus Clinical Professor of Surgery at the University of Colorado School of Medicine. In addition to his myriad other commitments, he served on a number of University committees.

During World War II he served with the 29th General Hospital in the South Pacific, and later at Fitzsimons General Hospital, Denver.

His passing leaves a void not only in organized medicine but also in the hearts and minds of his associates, friends, and patients.

He was married to Elizabeth Ann McAndrews July 5, 1927. They had three children, Kenneth C. Jr., MD, Robert B., MD, and Patricia Ann Sawyer Teets, all of whom survive the doctor along with nine grandchildren.

Colorado

Doctor Gordon W. Johnson, formerly of Clayton, New Mexico and Durango, Colorado, died in Denver, Tuesday April 12, 1977. He was 47 years of age. Born in Denver, July 31, 1929, he received a BA from the University of Denver, then his MD from the University of Colorado in 1957.

After an internship in Bernalillo County Indian Hospital, Albuquerque, New Mexico, he opened a family practice in Clayton, New Mexico. He moved to Durango, Colorado in 1967 to set up a practice, and remained there until forced by ill health to retire.

His widow, Shirley Ann, Lakewood, Colorado survives as do two sons, Gordon W. Johnson, Jr. and Charles E. Johnson, and two daughters, Kimberly Ann and Kristie Lynn, also surviving is his father, Charles E. Johnson of Durango, a brother Charles F. of Lakewood, and two sisters, Shirley Weise, Louisberg, Minnesota, and Betty Root, Belize, Central America.

Doctor Harry H. Nelson of Longmont, Colorado died on Monday, April 18, 1977. He was 77 years of age. Born September 8, 1899 at Lindentree, Ohio, he received his MD from Case Western Reserve University, Cleveland, Ohio in 1925. After an internship and six months of orthopaedic residency he developed pulmonary tuberculosis, and was sent to the National Jewish Hospital in Denver for treatment.

Following his recovery, he was an officer in the U.S. Public Health Service until he opened a family practice in Longmont in 1938. He was awarded a Fifty Year Medal of Service by the Colorado Medical Society in 1975.

He is survived by his wife, Helen, Longmont, Colorado, a daughter, Dr. Nancy Nelson, a Denver pediatrician, two sons, Dr. James Nelson, a pediatrician with the Army at Fort Lewis, Washington, and Doctor William Nelson, a psychiatrist in Raleigh, North Carolina. Also surviving are two grandsons, James and Mark, and a sister, Mrs. Helen James, Alliance, Ohio.

Doctor J. David Taylor of Colorado Springs, Colorado died Tuesday, May 3, 1977. Born September 27, 1918 in Prarieburg, Iowa, he received his MD from Bowman-Gray Medical School in North Carolina in 1945. After an internship at Winston-Salem Baptist Hospital, North Carolina, he entered the Air Force as flight surgeon. He left the service in 1949, taking a position at the Kansas State Tuberculosis Sanitorium in Norton, Kansas. In 1949 he opened a practice in Colorado Springs where he remained until his death.

His widow, Mrs. Alma Taylor, a daughter, Penny Taylor of Los Angeles, three brothers, Lt. General Richard Taylor, Washington, D.C.; Dr. Thomas Taylor, Salina, Kansas, and Phillip Taylor, Holyoke, Massachusetts, and three sisters, Mrs. Charlotte Shepherd, Norton, Kansas; Mrs. Lou Catalina, Wilmington, Delaware, and Miss Marguerite Taylor, Concord, California, also survive.

Doctor Demetrio A. Chaves, Denver, died March 26, 1977. Born in June 1920, in Magdalena, New Mexico, and educated in Albuquerque, he received his MD from the University of Colorado in 1949. Following an internship at the Bridgeport General Hospital, Bridgeport, Connecticut, he opened an office for the practice of general medicine and surgery in Antonito, Colorado.

In the mid-1950's he moved to Denver to open an office at 1633 Fillmore Street where he practiced until his death.

Doctor James Alex Johnson of Colorado Springs, Colorado died April 28, 1977 of injuries sustained in an auto accident while attending a medical meeting in Ireland.

Born in Empire, Michigan, April 20, 1917, he received his MD from the University of Michigan in 1943. After an internship at the University Hospital in Ann Arbor, Michigan, he joined the Navy and served until 1946 when he became a resident in Orthopaedic Surgery at the University of Michigan Hospital.

Upon completion of his training he opened a practice in Orthopaedic Surgery in Colorado Springs. He was certified in Orthopaedics in 1951. In addition to his membership in his county, state, and the American Medical Association, he held memberships in the American Academy of Orthopaedic Surgeons, the Clinical Orthopaedic Society, the Mid-Central and Rocky Mountain Orthopaedic societies, and the Western Orthopaedic Association.

He is survived by his wife Nancy, four daughters, Mary Richardson, Spokane, Washington; Katherine McDaniel, Chicago, Illinois; Cornelia Johnson, Vail, Colorado; Sarah Johnson, Canon City, Colorado, and a son, James Dunn Johnson, Colorado Springs.

Montana

Doctor Bob E. Hulit of Billings, Montana, died June 12, 1977 at the age of 59. Doctor Hulit had practiced in Billings since 1954. He received his MD from the University of Louisville School of Medicine in 1943, and received his license in 1953. He was a member of the American Board of Obstetrics and Gynecology, and of the American College of Obstetricians and Gynecologists, and of the American College of Surgeons.

Doctor Hulit was a member of the Montana and American medical associations, and of the Yellowstone Valley Medical Society. He is survived by his widow, two sons, two daughters, his mother, two brothers, a sister, and one grandchild.

THE BOOK CORNER

New books received are acknowledged in this section and such acknowledgment must be regarded as sufficient return for the courtesy of the sender. Selection will be made for review in the interests of our readers and as space permits. Books are listed with advance data supplied by publishers. Prices quoted are not guaranteed. For further information, address queries to the publishers. Books here listed are available for lending from the Denver Medical Society Library.

Denison Memorial Library at the University of Colorado Medical Center has announced new on-line search charges as of July 1. The rate for Medline (current file) will be $7.00 and Medline (backfiles) will be $7.50, as will Toxline. Other data bases (Psychological Abstracts, Biological Abstracts, Chemical Abstracts, etc.) will be $11.00.

Recent Acquisitions

BASELINES FOR SETTING HEALTH GOALS AND STANDARDS: U.S. Department of Health, Education and Welfare. Washington, D.C., G.P.O., 1976. 110 p. Gift. $1.55.

BEYOND A NEW PERSPECTIVE: Marc LaLonde. Miami Beach, Florida, American Public Health Association, 1976. 43 p. Gift.

BOTULISM: Louis Smith. Springfield, Thomas, 1977. 236 p. Review.

BRITISH NATIONAL HEALTH SERVICE COMPLAINTS PROCEDURES: Alonza S. Yerby. Bethesda, Md., U.S. Department of Health, Education and Welfare, National Institutes of Health, 1975. 65 p. Gift.

CLINICAL GASTROENTEROLOGY: Howard M. Spiro. 2nd ed. New York, Macmillan, 1977. 1289 p. $45.00.

COLPOSCOPY: Santiago Dexeus, et al. Philadelphia, Saunders, 1977. 266 p. (Major problems in obstetrics and gynecology, vol. 10)

DAVIS-CHRISTOPHER TEXTBOOK OF SURGERY: David C. Sabiston, ed. 11th ed. Philadelphia, Saunders, 1972. 2465 p. $35.00.

DIAGNOSIS OF SPEECH AND LANGUAGE DISORDERS: James E. Nation. St. Louis, Mosby, 1977. 453 p. $14.50.

DIAGNOSTIC ELECTROCARDIOGRAPHY AND VECTORCARDIOGRAPHY: Henry H. Friedman. 2nd ed. New York, McGraw-Hill, 1977.

DISTURBED CHILDREN AND THEIR FAMILIES: Alfred P. French. New York, Human Sciences Press, 1977. $19.95.

DOCTORS AND PATIENTS: Robert S. Smith. Boise, Idaho, Syms-York, 1976. 163 p. Gift. $8.00.

ESSENTIAL CONCEPTS OF CLINICAL PHYSIOLOGY: James V Lawry. Sunderland, Mass., Sinauer Associates, 1977. 246 p. Gift.

FOREIGN TRAVEL IMMUNIZATION GUIDE: Hans H. Neumann. 6th ed. Oradell, N.J., Medical Economics, 1977. Gift.

IMMUNOLOGY OF THE GUT: Symposium on Immunology of the Gut, London, 1976. New York, Elsevier, 1977. Gift.

INFORMATION TO AUTHORS; EDITORIAL GUIDELINES REPRINTED FROM 140 MEDICAL JOURNALS: Harrier R. Meiss and D.A. Jaeger, comp. New York, Mt. Sinai School of Medicine, 1976. 300 p $13.50.

MAN AT HIGH ALTITUDE: Donald Heath and David R. Williams. New York, Churchill Livingstone, 1977. Review. $28.00.

MEDICAL AND HEALTH ANNUAL: 1977: Encyclopaedia Britannica, Inc. Chicago, Encyclopaedia Britannica, 1977. Gift.

METABOLIC DISEASES OF BONE: Jenifer Jowsey. Philadelphia, Saunders, 1977. 312 p. (Saunders Monographs in Clinical Orthopaedics, vol. 1) $22.50.

MODERN DRUG ENCYCLOPAEDIA AND THERAPEUTIC INDEX: Arthur J. Lewis, ed. 14th ed. New York, Yorke Medical, 1977. 1009 p. $34.00.

THE NATIONAL HEALTH SYSTEM IN DENMARK: Dorte Gannik. Bethesda, Md., U.S. Department of Health, Education and Welfare, Public Health Service, National Institutes of Health, 1976. Gift. $1.15.

OTOLARYNGOLOGY: Gerald M. English. New York, Harper & Row, 1976. 815 p. $42.50.

PHYSICIANS' GUIDE TO MEDICAL ADVICE FOR OVERSEAS TRAVELERS: American Medical Association. Chicago, A.M.A., 1973. 23 p. Gift.

PUBLIC HEALTH PLANNING IN THE U.S.S.R.: Bethesda, MD., U.S. Department of Health, Education and Welfare, Public Health Service, National Institutes of Health, 1976. 273 p. Gift.

THE RESTORATION OF THE SELF: Heinz Kohut. New York, International Universities Press, 1977. 345 p. $17.50.

SELECTED COMMUNITY HOSPITAL INDICATORS: 1975. American Hospital Association. Chicago, A.H.A., 1975.

THEORETICAL PRINCIPLES OF PSYCHOSOMATIC MEDICINE: Ivan T. Kurtsin. New York, Wiley, 1976. 257 p. $35.00.

TUMOURS OF THE OVARY: H. Fox and F.A. Langley. Chicago Year Book, 1976. 312 p. $20.00.

STANFORD UNIVERSITY SCHOOL OF MEDICINE
OFFICE OF POSTGRADUATE MEDICAL EDUCATION

announces six multidisciplinary courses for 1977-78

These week-long continuing education courses are designed to offer the practicing physician an opportunity to assess present knowledge and practice in important areas of medicine, and to acquire new information and skills of practical use.

Each course consists of lectures covering topics of general interest and a series of elective problem-solving sessions, seminars and demonstrations dealing in depth with specialized topics.

A large number of Stanford University School of Medicine faculty members, from various clinical and basic science departments, will take part in each of these programs.

Primary Care

October 3-7, 1977, at Stanford

Designed for family and general practitioners, general internists, and general pediatricians, this meeting will deal with the management of patients with problems commonly encountered in primary care. Tuition: $265.

Intensive Care

October 31-November 4, 1977, at Stanford

This program will review the current status of management principles and procedures applicable to critically ill and critically injured patients. Tuition: $265.

Therapy: Principles and Use of Drugs

January 23-27, 1978, at Stanford

This course, suitable for all physicians, will be an intensive review of the modern concepts of drug action and of the practical aspects of the use of a large variety of pharmacologic agents. Tuition: $265.

Basic Science for Clinicians

February 27-March 3, 1978, at Stanford

This comprehensive review of basic science is designed to expand the clinician's knowledge of molecular biology. Tuition: $265.

Cancer

March 27-31, 1978, at Stanford

The present knowledge of the pathogenesis of cancer and clinical aspects of diagnosis, classification, staging, and management of malignant disorders will be reviewed in this course, which is intended for all physicians and surgeons who deal with problems related to neoplastic disease. Tuition: $265.

Management of the Surgical Patient

April 29-May 6, 1978, at Mauna Kea Beach Hotel, Kamuela, Hawaii

This course is designed for all physicians—both surgeons and nonsurgeons—who participate in the care of surgical patients. Tuition: $275; room deposit: $125, payable to Stanford.

The Postgraduate Medical Education program at Stanford University School of Medicine is fully accredited by the Council on Medical Education of the American Medical Association and approved for Category I Credit toward the California Medical Association's Certificate in Continuing Education. Attendance certificates are provided.

EARLY APPLICATION IS ADVISED — USE COUPON BELOW

Clip and mail to
OFFICE OF POSTGRADUATE MEDICAL EDUCATION
Stanford University School of Medicine, Room TA 145, Stanford, California 94305 / Telephone (415) 497-5594

ENROLL ME IN ____**Primary Care** ($265 enclosed); ____**Intensive Care** ($265 encolsed); ____**Therapy** ($265 enclosed); ____**Basic Science** ($265 enclosed); ____**Cancer** ($265 enclosed); ____**Hawaii Course** ($400 enclosed)

SEND BROCHURES ON ____Primary Care: ____Intensive Care; ____Therapy; ____Basic Science; ____Cancer; ____Hawaii Course

NAME_____Specialty_____
Last First (please print)

ADDRESS_____

_____ZIP_____Daytime Phone_____

(Check payable to STANFORD UNIVERSITY SCHOOL OF MEDICINE)

MEETINGS

Colorado Medical Society
107TH ANNUAL MEETING

The Broadmoor, Colorado Springs, Colorado
September 21-25, 1977

University of Colorado School of Medicine
Continuing Medical Education

2ND ANNUAL SYMPOSIUM ON VASCULAR SURGERY
The Marriott Hotel, Denver, Colorado

September 29-October 1, 1977
Credit: Approved for 20 hours of AMA Category 1

* * *

TREATMENT OF THE ABUSED AND NEGLECTED CHILD: PART II
The Executive Tower Inn, Denver, Colorado

October 6-8, 1977
Credit: Approved for AMA Category 1; prescribed credit for AAFP applied for.

* * *

LUNG DISEASE IN CHILDREN
THE MARK, Vail, Colorado

January 8-11, 1978
Credit: Approved for Category 1

* * *

FOURTH ANNUAL WINTER SKIN SEMINAR
The Given Institute of Pathobiology, Aspen, Colorado

February 12-17, 1978
Credit: Approved for AMA Category 1, prescribed credit for AAD
Contact: Office of Postgraduate Medical Education, University of Colorado Medical Center, Container C295, 4200 East 9th Avenue, Denver, Colorado 80262.

The American Society for Parenteral and Enteral Nutrition (ASPEN)
HYPERALIMENTATION IN MODERN HOSPITAL PRACTICE

Surgical Grand Rounds
Conference Room, Presbyterian Medical Center, Denver

September 29, 1977, 8:00 am - 9:30 am

* * *

Repeat of Program
September 29, 1977, 12:00-1:30 pm
St. Joseph Hospital, Denver
Contact: Michael Lubchenco, MD, (303) 534-9011
Credit: 2 hours category 1 CME for Physicians Recognition Award

Montana Medical Association
99TH ANNUAL MEETING

Village Motor Inn, Missoula, Montana
September 29-October 1, 1977

Montrose Memorial Hospital
6TH ANNUAL FALL CLINICS

Elk's Lodge, 415 Hillcrest Drive,
Montrose Colorado
September 30-October 1, 1977
Credit: AAFP, ACGP 10 credit hours approved.
Contact: Mr. Stephen T. Scott, Montrose Memorial Hospital, Montrose, Colorado 81401

American Association of Electromyography and Electrodiagnosis
24TH ANNUAL MEETING

Hilton Hotel, Salt Lake City, Utah

September 30-October 1, 1977
Contact: Michael Cherington, MD, University of Colorado Medical Center, Department of Neurology, 4200 East 9th Avenue, Denver, Colorado 80220
Credit; 9 hours of AMA - approved CME

OUTPATIENT AND INPATIENT MANAGEMENT OF ASTHMA AND COPD
Weld County General Hospital Auditorium, Greeley, Colorado

October 1, 1977
Credit: CME 4 hours Category 1
Contact: Robert Cash, MD, 1900 16th Street, Greeley, Colorado 80631 or Call: (303) 353-1551, Ex. 289.

Colorado Association for Continuing Medical Laboratory Education, Inc.
CLINICAL AND LABORATORY DIAGNOSIS OF INFECTIOUS DISEASE

Dawson Hall, Presbyterian Medical Center, 1719 East 19th Avenue, Denver, Colorado

October 4, 7-10 pm - Immediate Aids in the Diagnosis of Infectious Disease, John Matsen, MD, Thomas Merrick, MD

October 11, 7-10 pm - Meningitis, Donald Kearns, MD, James Todd, MD

October 18, 7-10 pm - Pneumonia and Respiratory Infections, Robert Van Scoy, MD, Elmer W. Koneman, MD

November 1, 7-10 pm - Infections Caused by Viruses, Chlamydia and Mycoplasma, Kenneth McIntosh, MD, Thomas Smith, MD

November 8, 7-10 pm - Subacute Bacterial Endocarditis and Septicemia Kenneth Ryan, MD, Barth Reller, MD

November 17, 7-10 pm - Anaerobic Infections, Sidney Finegold, MD, Stephen Allen, MD

Credit: 1.8 CEU's - entire series; 0.3 CEU's individual courses. Approved category I AMA-ASCP.
Contact: CACMLE, Mercy South, Room 520, 1601 Milwaukee St., Denver, Colorado 80206

American Academy of Occupational Medicine - American Academy of Industrial Hygiene
JOINT CONFERENCE ON OCCUPATIONAL HEALTH

Brown Palace Hotel, Denver, Colorado
October 5-7, 1977

Contact: American Academy of Occupational Medicine, 150 N. Wacker Drive, Chicago, Illinois 60606

Credit: 10.5 hours credit Category 1 AMA Physicians Recognition Award; 10 prescribed hours credit AAFP

American Cancer Society, Montana Division
ANNUAL MEETING

Northern Hotel, Billings, Montana
October 7-8, 1977

Contact: Mrs. Ruth Eaton, 2820 1st Avenue South, Billings, Montana, 59101

American Academy of Family Physicians
ANNUAL SCIENTIFIC ASSEMBLY
Convention Center, Las Vegas, Nevada

October 10-13, 1977

Contact: William R. DeLay, Phone: (800-821-2512)

Credit: One hour prescribed AAFP CME for each hour of participation in scientific programs.

American College of Cardiology
THE CLINICAL SPECTRUM OF ADULT HEART DISEASE

October 13-15, 1977
Basic Medical Sciences Building
University of New Mexico Medical Center
Albuquerque, New Mexico

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10TH ANNUAL CARDIOVASCULAR SYMPOSIUM
Americana Hotel, New York City, New York December 9-11, 1977

Credit: 23 hours Category I AMA, AAFP.

* * *

9TH ANNUAL CARDIOVASCULAR CONFERENCE AT SNOWMASS
Snowmass Resort, Snowmass, Colorado

January 9-13, 1978

Contact: Mary Anne McInerny, Director, Division of Continuing Education, American College of Cardiology, 9650 Rockville Pike, Bethesda, Maryland 20014

* * *

COLORADO ASSOCIATION FOR CONTINUING MEDICAL LABORATORY EDUCATION, INC.

Courses for Physicians AMA Category A-1 Credit (ASCP Approved)

MICROBIOLOGY: *Clinical and Laboratory Diagnosis of Infectious Disease. October and November (See RMMJ Meetings)*

Faculty:

Stephen D. Allen, MD	*Thomas M. Merrick, MD*
Sidney M. Finegold, MD	*L. Barth Reller, MD*
Donald Kearns, MD	*Kenneth J. Ryan, MD*
Elmer W. Koneman, MD	*Thomas A. Smith, PhD*
Kenneth McIntosh, MD	*James Todd, MD*
John M. Matsen, MD	*Robert E. Van Scoy, MD*

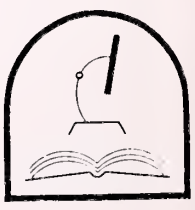
COAGULATION: Thromboembolic Disorders, October 10
Peter Steele, MD
The Surgeon and the Bleeding Patient, A Practical Approach, October 19 and 20. *Ben Galloway, MD,* and *Lee Gates, MD*
The Bleeding Patient, November 15. *Sanford D. Peck, MD*

HEMATOLOGY: Thalassemia Syndromes and Hemoglobinopathies. November 8, *John B. Miale, MD*

IMMUNOLOGY: Clinical Immunology, November 16, *Leo P. Cawley, MD*

For further details and course outlines, call or write:

CACMLE, INC. 1601 Milwaukee Street
Denver, Colorado 80206 (303) 321-1734



Lovelace-Bataan Medical Center
RECENT ADVANCES IN OBSTETRICS AND GYNECOLOGY

October 21-22, 1977
Airport Marina Hotel, Albuquerque, New Mexico
NEW MEXICO METABOLISM SYMPOSIUM

* * *

March 9-11, 1978
Taos, New Mexico

Contact: Office of Medical Education, Lovelace Foundation for Medical Education and Research, 5200 Gibson Blvd., SE, Albuquerque, New Mexico, 87108.
Credit: AAFP approval applied for. Category 1 AMA : Physician's Recognition Award.

Pacific Medical Center
LIVING WITH CHRONIC AND TERMINAL ILLNESS

October 27-28, 1977

Presbyterian Hospital, Pacific Medical Center, Clay and Buchanan Streets, San Francisco, California, 94120

American College of Chest Physicians
43RD ANNUAL SCIENTIFIC ASSEMBLY

October 30-November 3, 1977
MGM Grand Hotel, Las Vegas, Nevada
Contact: American College of Chest Physicians, 911 Busse Highway, Park Ridge, Illinois 60068

Institute for Professional Educational Development
A COMPREHENSIVE CLINICAL HYPNOSIS AND BEHAVIOR THERAPY CONFERENCE

November 4-6, 1977
Brown Palace Hotel, Denver, Colorado
Credit: Approved AMA Category 1, 21 credit hours
Contact: Institute for Professional Educational Development, 250 Arapahoe Avenue, Boulder, Colorado, 80302

Penrose Hospital Department of Pathology
PENROSE DIAGNOSTIC CONFERENCE

The Broadmoor, Colorado Springs, Colorado

November 12, 1977
Contact: Penrose Diagnostic Conference, Pathologoy Department, Penrose Hospital, Colorado Springs, Colorado

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FOR SALE

1800 Downing Street Medical Building
(Across from Children's Hospital)

5000 square foot dental office
can be used for other medical offices
Excellent terms available to qualified buyer

Beverly Parks

The Trowbridge Agency

(303) 534-7640
Specializing in Real Estate in the Midtown
Denver Medical Area

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A Comprehensive Clinical Hypnosis and Behavior Therapy Conference

Sponsor: INSTITUTE FOR PROFESSIONAL EDUCATIONAL DEVELOPMENT

Conducted by:
Presley Reed, M.D.
Ira Pearlman, Ph.D
Rita Zimbelman, R.N., M.S.

November 4-6, 1977

The Brown Palace Hotel
17th & Tremont Place
Denver, Colorado

Approved: AMA Category I, 21 Credit Hrs.

This conference is designed to provide participants with an intensive course of instruction in combining hypnosis and behavioral techniques. Contact: IPED, 250 Arapahoe Avenue, Boulder, Colorado, 80302. Phone: (303) 443-4734

American Medical Women's Association
62ND ANNUAL MEETING

The Marriott Hotel, Denver, Colorado

November 30-December 4, 1977

Credit: Approved for Category 1, AMA - 10 credit hours,. Approved for Category 1, AAFP.
Contact: American Women's Medical Association, 1740 Broadway, New York, New York, 10019

Genetic and Birth Defects Unit, Shodair Hospital, Helena, Montana
SECOND ETHICS—MORALS SEMINAR ON GENETICS

Carroll College, Helena, Montana
December 1-2, 1977

Credit: AMA Category I
Contact: Philip D. Pallister, MD, Shodair Hospital, P.O. Box 1715, Helena, Montana 59601

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General Psychological Principles for Primary Care Physicians: October 4 - 7, 1977. An overview.

Psychological Medications for Primary Care Physicians: November 11-12, 1977. An overview of therapeutic uses and side effects of psychoactive medications. Seminar leader is Robert Obourn, M.D.

Alcoholism: Pathways to Understanding and Recovery: November 18 - 19, 1977. Workshop will discuss early identification, non-judgmental confrontation and sources for referral for your patients and their families. George Penn, M.D., will be the seminar leader.

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All workshops will be in Topeka, Kansas, at The Menninger Foundation. For further information contact: June Housholder, Division of Continuing Education, The Menninger Foundation, P.O. Box 829, Topeka, Kansas 66601. 913-234-9566, ext. 3685.

American College of Physicians
NEW MEXICO REGIONAL MEETING

George Savage Auditorium, Presbyterian Hospital Center, Albuquerque, New Mexico

December 2-3, 1977
Contact: Fred H. Hanold, MD, 1421 Dartmouth NE, Albuquerque, New Mexico 87106.

* * *

COLORADO REGIONAL MEETING

The Broadmoor, Colorado Springs, Colorado

January 12-14, 1978
Contact: Edward S. Miller, MD, 1050 Clermont St., Denver, Colorado 80220

American Medical Association
31ST ANNUAL CLINICAL CONVENTION

Palmer House, Chicago, Illinois

December 4-7, 1977

West Coast Allergy Society
16TH ANNUAL SCIENTIFIC SESSION

Camelback Inn, Scottsdale, Arizona

December 14-17, 1977
Contact: Robert L. Cutter, MD, 5210 S.W. Corbett Avenue, Portland, Oregon 97201

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For further information contact:

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2650 Yale Blvd., SE Suite 103
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Rocky Mountain Medical Journal

November-December 1977
Volume 74 - Number 6

ROCKY MOUNTAIN MEDICAL JOURNAL
NOV. - DEC. 1977

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Not recommended for use in persons under 15 years of age. Though physical and psychological dependence have not been reported on recommended doses, use caution in administering to addiction-prone individuals or those who might increase dosage.

Precautions: In elderly and debilitated, limit initial dosage to 15 mg to preclude oversedation, dizziness and/or ataxia. Consider potential additive effects with other hypnotics or CNS depressants. Employ usual precautions in patients who are severely depressed, or with latent depression or suicidal tendencies. Periodic blood counts and liver and kidney function tests are advised during repeated therapy. Observe usual precautions in presence of impaired renal or hepatic function.

Adverse Reactions: Dizziness, drowsiness, lightheadedness, staggering, ataxia and falling have occurred, particularly in elderly or debilitated patients. Severe sedation, lethargy, disorientation and coma, probably indicative of drug intolerance or overdosage, have been reported. Also reported: headache, heartburn, upset stomach, nausea, vomiting, diarrhea, constipation, GI pain, nervousness, talkativeness, apprehension, irritability, weakness, palpitations, chest pains, body and joint pains and GU complaints. There have also been rare occurrences of leukopenia, granulocytopenia, sweating, flushes, difficulty in focusing, blurred vision, burning eyes, faintness, hypotension, shortness of breath, pruritus, skin rash, dry mouth, bitter taste, excessive salivation, anorexia, euphoria, depression, slurred speech, confusion, restlessness, hallucinations, paradoxical reactions, *e.g.,* excitement, stimulation and hyperactivity, and elevated SGOT, SGPT, total and direct bilirubins and alkaline phosphatase.

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References:

1. Kales A, *et al*: *Clin Pharmacol Ther 20*: 541-545, Nov 1976
2. Kales A, *et al*: *Clin Pharmacol Ther 19*:576-583, May 1976
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4. Dement WC, Guilleminault C, Zarcone V: Progress in clinical sleep research. Scientific exhibit at the American Medical Association, Atlantic City NJ, Jun 14-18, 1975
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7. Moore JD, Weissman L: *J Clin Pharmacol 16*:241-244, May-Jun 1976
8. Spiegel HE: Data on file, Medical Department, Hoffmann-La Roche Inc., Nutley NJ
9. Kaplan SA, *et al*: *J Pharm Sci 62*:1932-1935, 1973

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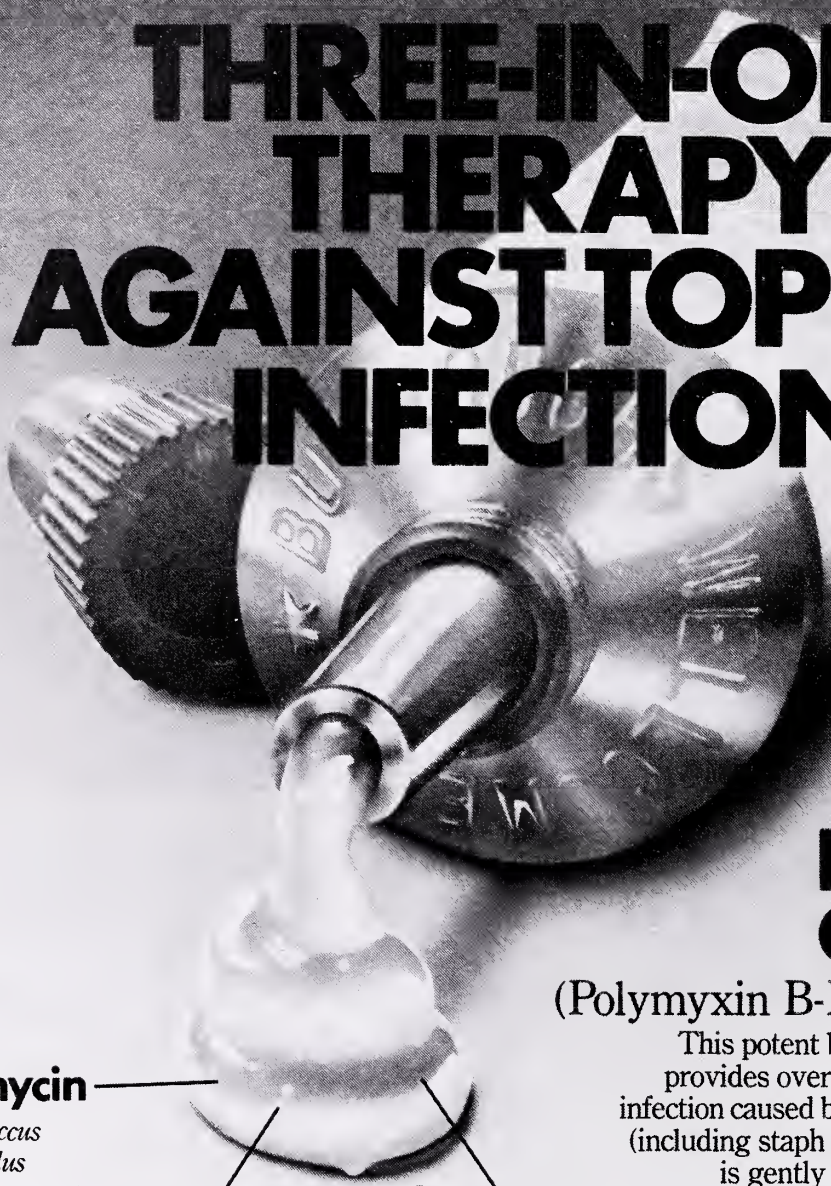

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Rocky Mountain Medical Journal

COLORADO
MONTANA
NEW MEXICO
WYOMING

November-December 1977
VOLUME 74 • NUMBER 6

OUR COVER: Sand castles of The Gods, the fantastic 23 labyrinthian miles of The Carlsbad Caverns National Park in southern New Mexico with their stalactites (they hang) and stalagmites (they rise) are a geologic wonder of the entire world; they comprise the largest known cave in the world. Water running through and dripping on limestone has turned these passageways into a great tourist attraction, and astonishment has led people to return again and again. A natural pride of New Mexico. Credit: New Mexico Tourist Bureau.

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MEETINGS

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62ND ANNUAL MEETING

The Marriott Hotel, Denver, Colorado

November 30-December 4, 1977

Credit: Approved for Category 1, AMA - 10 credit hours,. Approved for Category 1, AAFP.
Contact: American Women's Medical Association, 1740 Broadway, New York, New York, 10019

Genetic and Birth Defects Unit, Shodair Hospital, Helena, Montana
SECOND ETHICS—MORALS SEMINAR ON GENETICS

Carroll College, Helena, Montana
December 1-2, 1977

Credit: AMA Category I
Contact: Philip D. Pallister, MD, Shodair Hospital, P.O. Box 1715, Helena, Montana 59601

American College of Physicians
NEW MEXICO REGIONAL MEETING

George Savage Auditorium, Presbyterian Hospital Center, Albuquerque, New Mexico

December 2-3, 1977
Contact: Fred H. Hanold, MD, 1421 Dartmouth NE, Albuquerque, New Mexico 87106.

* * *

COLORADO REGIONAL MEETING

In conjunction with Colorado Society of Internal Medicine

The Broadmoor, Colorado Springs, Colorado

January 12-14, 1978
Contact: Edward S. Miller, MD, 1050 Clermont St., Denver, Colorado 80220

* * *

CLINICAL ONCOLOGY FOR THE GENERAL INTERNIST
Tamaron, Durango, Colorado

February 28-March 2, 1978
Credit: 18 hours Category 1 CME
Contact: Registrar, American College of Physicians, P.O. Box 7777 R0510, Philadelphia, Pennsylvania, 19175

American Medical Association
31ST ANNUAL CLINICAL CONVENTION

Palmer House, Chicago, Illinois

December 4-7, 1977

American College of Cardiology

10TH ANNUAL CARDIOVASCULAR SYMPOSIUM
Americana Hotel, New York City, New York December 9-11, 1977
Credit: 23 hours Category I AMA, AAFP.

* * *

9TH ANNUAL CARDIOVASCULAR CONFERENCE AT SNOWMASS
Snowmass Resort, Snowmass, Colorado

January 9-13, 1978

Contact: Mary Anne McInerny, Director, Division of Continuing Education, American College of Cardiology, 9650 Rockville Pike, Bethesda, Maryland 20014

West Coast Allergy Society
16TH ANNUAL SCIENTIFIC SESSION

Camelback Inn, Scottsdale, Arizona

December 14-17, 1977
Contact: Robert L. Cutter, MD, 5210 S.W. Corbett Avenue, Portland, Oregon 97201

University of Colorado School of Medicine
Continuing Medical Education

LUNG DISEASE IN CHILDREN
THE MARK, Vail, Colorado

January 8-11, 1978
Credit: Approved for Category 1

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PEDIATRIC POSTGRADUATE COURSE
THE MARK, Vail, Colorado

January 11-14, 1978
Credit: Approved for 18 hours, AMA Category 1

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HORIZONS IN SURGERY
THE MARK, Vail, Colorado

January 15-20, 1978
Credit: Approved for AMA Category 1

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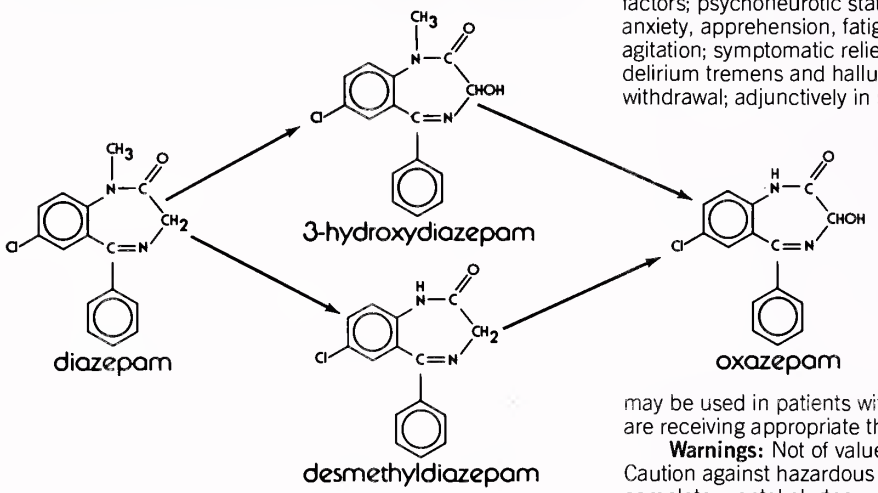
A pharmacokinetic character all its own



Valium (diazepam) is a benzodiazepine with a distinctive pharmacokinetic profile

The pharmacokinetic profile of Valium is one of the characteristics that sets it apart from other benzodiazepines. Consider, in particular, the metabolic pathway of Valium. The three major metabolites of Valium exhibit significant pharmacologic activity—and so, of course, does the parent substance—diazepam itself. All combine to produce the characteristic clinical response seen with Valium. The response you have come to know, to want and to trust.

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Valium® (diazepam) IV

2-mg, 5-mg, 10-mg scored tablets

a prudent choice in psychic tension and anxiety

Before prescribing, please consult complete product information, a summary of which follows:

Indications: Tension and anxiety states; somatic complaints which are concomitants of emotional factors; psychoneurotic states manifested by tension, anxiety, apprehension, fatigue, depressive symptoms or agitation; symptomatic relief of acute agitation, tremor, delirium tremens and hallucinosis due to acute alcohol withdrawal; adjunctively in skeletal muscle spasm due to reflex spasm to local pathology; spasticity caused by upper motor neuron disorders; athetosis; stiff-man syndrome; convulsive disorders (not for sole therapy).

Contraindicated: Known hypersensitivity to the drug. Children under 6 months of age. Acute narrow angle glaucoma; may be used in patients with open angle glaucoma who are receiving appropriate therapy.

Warnings: Not of value in psychotic patients. Caution against hazardous occupations requiring complete mental alertness. When used adjunctively in convulsive disorders, possibility of increase in frequency and/or severity of grand mal seizures may require increased dosage of standard anticonvulsant medication; abrupt withdrawal may be associated with temporary increase in frequency and/or severity of seizures. Advise against simultaneous ingestion of alcohol and other CNS depressants. Withdrawal symptoms (similar to those with barbiturates and alcohol) have occurred following abrupt discontinuance (convulsions, tremor, abdominal and muscle cramps, vomiting and sweating). Keep addiction-prone individuals under careful surveillance because of their predisposition to habituation and dependence.

Usage in Pregnancy: **Use of minor tranquilizers during first trimester should almost always be avoided because of increased risk of congenital malformations as suggested in several studies. Consider possibility of pregnancy when instituting therapy; advise patients to discuss therapy if they intend to or do become pregnant.**

Precautions: If combined with other psychotropics or anticonvulsants, consider carefully pharmacology of agents employed; drugs such as phenothiazines, narcotics, barbiturates, MAO inhibitors and other antidepressants may potentiate its action. Usual precautions indicated in patients severely depressed, or with latent depression, or with suicidal tendencies. Observe usual precautions in impaired renal or hepatic function. Limit dosage to smallest effective amount in elderly and debilitated to preclude ataxia or oversedation.

Side Effects: Drowsiness, confusion, diplopia, hypotension, changes in libido, nausea, fatigue, depression, dysarthria, jaundice, skin rash, ataxia, constipation, headache, incontinence, changes in salivation, slurred speech, tremor, vertigo, urinary retention, blurred vision. Paradoxical reactions such as acute hyperexcited states, anxiety, hallucinations, increased muscle spasticity, insomnia, rage, sleep disturbances, stimulation have been reported; should these occur, discontinue drug. Isolated reports of neutropenia, jaundice; periodic blood counts and liver function tests advisable during long-term therapy.

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Donnagel with paregoric equivalent

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Kaolin	6.0 g.
Pectin	142.8 mg.
Hyoscyamine sulfate	0.1037 mg.
Atropine sulfate	0.0194 mg.
Hyoscine hydrobromide	0.0065 mg.
Powdered opium, USP (equivalent to paregoric 6 ml.) (warning: may be habit forming)	24.0 mg.
Sodium benzoate (preservative)	60.0 mg.
Alcohol, 5%	

Now with child-proof closure

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Richmond, Virginia 23220

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EXPECTORANT
NASAL DECONGESTANT
ANTITUSSIVE
16 FL.OZ.(1PT.)
THIS PACKAGE INTENDED FOR REPACKAGING BY PHARMACIST
630

A·H·ROBINS

INTRODUCING...

NEW ROBITUSSIN®-DAC

for the difficult cough complicated by nasal congestion

*To help clear the lower respiratory tract—the most recommended single expectorant in the U.S.**

Guaifenesin, NF . . . . . 100 mg/5 ml

- Enhances output of less viscid secretions to aid in the removal of inspissated mucus.
- Relieves irritated membranes in respiratory passageways by preventing dryness through increased mucus flow.
- Makes dry, unproductive coughs more productive and less frequent.

For cough suppression...the drug of choice†—

Codeine Phosphate, USP . . . . . 10 mg/5 ml

(Warning: May be habit forming.)

- Reduces severity, frequency and patient awareness of cough.
- Promotes patient comfort.
- Low drug dependence and little risk of side effects in recommended dosage.

For accompanying nasal congestion—

Pseudoephedrine HCl, NF . . . . . 30 mg/5 ml

- An orally effective nasal/sinus decongestant.
- Relieves congestion, reduces edema, promotes drainage.
- 60 mg pseudoephedrine in a 2-teaspoonful adult dose.

Available in pints only. You prescribe the quantity dispensed.

References:
*National Disease & Therapeutic Index, Jan.-Dec., 1976. IMS America Ltd., Ambler, Pa. 1976.
†Amer. Med. Assn., Dept. of Drugs, A.M.A. Drug Evaluations, 2nd Edition, Publishing Sciences Group, Inc., Acton, Mass., 1973, pp. 482-3.

Robitussin®-DAC—Each 5 ml (1 teaspoonful) contains: Guaifenesin, NF 100 mg; Pseudoephedrine Hydrochloride, NF 30 mg; Codeine Phosphate, USP 10 mg (Warning: May be habit forming) in a palatable, aromatic syrup. Alcohol, 1.4 per cent. INDICATIONS: For the temporary relief of cough and nasal congestion as may occur with the common cold or with inhaled irritants. CONTRAINDICATIONS: Hypersensitivity to any of the ingredients, marked hypertension, hyperthyroidism, or in patients who are receiving MAO inhibitors or antihypertensive medication. WARNINGS: Use this product with caution in children under 2 years or in children taking another drug. Prescribe cautiously for patients with persistent or chronic cough such as comes with smoking, asthma, emphysema, or where cough is accompanied by excessive secretions. Caution should be taken in administering this drug to patients with high blood pressure, heart disease or diabetes. In patients with chronic pulmonary disease or shortness of breath, this product should be administered with caution. As with all products containing sympathomimetic amines, use with caution in patients with prostatic hypertrophy or glaucoma. Do not exceed recommended dosage because at higher doses nervousness, dizziness or sleeplessness may occur. May cause or aggravate constipation. NOTE: Guaifenesin has been shown to produce a color interference with certain clinical laboratory determinations of 5-hydroxyindoleacetic acid (5-HIAA) and vanillylmandelic acid (VMA). ADVERSE REACTIONS: Agitation, dizziness, insomnia, palpitations or nausea may occur. In such instances, reduction in frequency and/or quantity of dose is indicated. RECOMMENDED DOSAGE: Adults—1 or 2 teaspoonfuls every four hours, not to exceed 12 teaspoonfuls in a 24-hour period; children 6 to under 12 years: 1 teaspoonful every four hours, not to exceed 6 teaspoonfuls in a 24-hour period; children 2 to under 6 years: 1/2 teaspoonful every four hours, not to exceed 3 teaspoonfuls in a 24-hour period; use as directed by a physician. ALSO AVAILABLE: Robitussin®—Robitussin A-C®—Robitussin with codeine. Robitussin®-CF®—Robitussin with phenylpropanolamine and dextromethorphan. Robitussin-DM®—Robitussin-DM in solid form). Cough Calmers® lozenges (Robitussin-DM and dextromethorphan. Robitussin-PE®—Robitussin with pseudoephedrine. A. H. Robins Co., Richmond, Va. 23220.

OFFER—For a 1978 calendar featuring a poster-size, full-color reproduction of photo of Southern Railway Engine No. 4501 to "Train Offer," on your Rx pad "**Robitussin-DAC**" A. H. Robins Company, Cherwood Avenue, Richmond, VA 23220.

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Signs Certificate of Ratification at His Home Without Women Witnesses.

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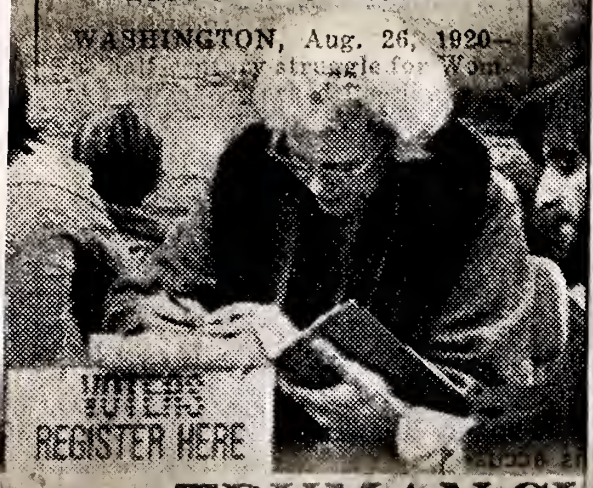
Wanted Movies of Ceremony, But Both Factions Are

WASHINGTON, Aug. 26, 1920—



Social Security Bill Is Signed; Gives Pensions to Aged, Job

Roosevelt Approves Message Intended to Benefit 30,0 Persons When States Adopt Cooperating Laws–He the Measure 'Cornerstone' of His Economic Progra

WASHINGTON, Aug. 14 The Social Security Bill, p a broad program of unemp insurance and old age p and counted upon to bene 20,000,000 persons, became day when it was signed b dent Roosevelt in the pre those chiefly responsible ting it through Congress.

Mr. Roosevelt called the "the cornerstone in a s which is being built but i means of stable

SENATE APPROVES 18-YEAR OLD VOTE IN ALL ELECTIONS

Amendment to Constitution is Sent to House, Where Passage is Expected

WASHINGTON, March 10, 1971—The Senate approved today, 94 to 0, and sent

TRUMAN CLOSES ITED NATIONS CONFEREN WITH PLEA TO TRANSLATE CHARTER INTO DEEDS

NEW WORLD HOPE

President Hails 'Great Instrument of Peace,' Insists It Be Used

HISTORIC LANDMARK

Meeting Gives Standing Ovation as Executive Pictures Peace Gain

"If we fail to use it," he declared to the solemn final meeting of the delegates, 'we shall betray all of those who have died in order that we might meet here in freedom and safety to create it.'

"If we seek to use it selfishly—for the advantage of any one nation or any small group of nations—we shall be equally guilty of that betrayal."

Fervent Interpolation

The President, speaking in the auditorium of the War Memorial Opera House, built in memory of sons of the Golden Gate city who gave their lives in the first World War, in which he himself served, seemed to give unconscious expression to the solemn feeling of the occasion when, at the outset of his speech, he interpolated the words, half a hope, half a prayer:

"Oh, what a great day this can be in history!"

Just before the plenary session

IGNI the Dra Ends No

WASHINGTON, Jan. 27, 1973—"With the signing of the peace agreement in Paris today, and after receiving a report from the Secretary of the Army that

PATIENT PACKAGE INSERTS: A CONCEPT WHOSE TIME HAS COME?

The consumer's right to know is an irreversible and desirable trend of the Seventies. It extends, and properly, to a patient's right to know more about his or her prescription medications. One way, gaining favor, is through patient package inserts. Wisely-prepared and properly distributed when medically indicated, they could markedly improve patient knowledge and drug therapy—laudable goals by anyone's standards.

The PMA endorses these goals and will work with government, the health professions and consumers to achieve them.

The Advantages

The concept holds promise of benefits: better patient understanding of the product prescribed, better adherence to the treatment plan, and more awareness of possible side reactions.

Every doctor has had patients who fail to finish antibiotic regimens because they feel better. Some patients assume that if one tranquilizer or analgesic is good, two may be twice as good. Still others fail to report dizziness while on antihypertensive therapy —and so on.

Problems like these might arise less often if the patient received written information in addition to verbal instructions. Some studies suggest that patients are more receptive to such materials, and they more often understand the verbal instructions and follow them, when inserts are used.

The Disadvantages

There are also some potential problems. Obviously, the inserts must be clearly phrased, without extraneous or complex detail. How much information is enough? How can it be kept current? Should all patients receive the same information? Should inserts be included with all drugs? Should only potential problems be listed or are patients better off with a "fair balance" presentation that describes usefulness as well as drawbacks?

These and similar questions require answers, since model inserts have yet to be properly developed and tested. Despite the need for these studies, the FDA is proceeding prematurely with inserts on selected products. We think the Congress is the only place where the matter can be given the proper legal status and direction, particularly since it represents a conceptual change in the legal, medical and social framework of the nation's prescription drug information system.

The Solution

The PMA believes that carefully-devised pilot studies of various kinds of inserts are needed. They should be developed and implemented with full participation by doctors, pharmacists, consumers, communications experts and the drug industry. Such studies will provide reliable pathways to follow, so that inserts will be useful aids to medical practice.

And particularly we think that you should be closely involved in this debate and in these studies and decisions. Otherwise, people with less experience and qualifications may control the purposes, content and use of a tool with considerable promise for improved patient care. It could make a difference in your practice tomorrow, and more importantly, in the health of your patients.

PMA
THE PHARMACEUTICAL MANUFACTURERS ASSOCIATION
1155 FIFTEENTH ST., N. W., WASHINGTON, D. C. 20005

B.W.CO.® HAS PUT MORE POTENCY IN THE LINE

EMPRACET® with Codeine Phosphate, 60 mg, No. 4 ©

EMPRACET® with Codeine Phosphate, 30 mg, No. 3 ©

CONTRAINDICATIONS: Hypersensitivity to acetaminophen or codeine.

WARNINGS: Drug dependence. Codeine can produce drug dependence of the morphine type and may be abused. Dependence and tolerance may develop upon repeated administration; prescribe and administer with same caution appropriate to oral narcotics. Subject to the Federal Controlled Substances Act.

Usage in ambulatory patients. Caution patients that these products may impair mental and/or physical abilities required for performance of potentially hazardous tasks such as driving a car or operating machinery.

Interaction with other CNS depressants. Patients receiving other narcotic analgesics, general anesthetics, phenothiazines, tranquilizers, sedative-hypnotics, or other CNS depressants (including alcohol) may exhibit additive CNS depression; when used together reduce dose of one or both.

Usage in Pregnancy. Safe use is not established. Should not be used in pregnant patients unless potential benefits outweigh possible hazards.

PRECAUTIONS: Head injury and increased intracranial pressure. Respiratory depressant effects of narcotics and their capacity to elevate cerebrospinal fluid pressure may be markedly exaggerated in the presence of head injury, other intracranial lesions or a pre-existing increase in intracranial pressure. Narcotics produce adverse reactions which may obscure the clinical course of patients with head injuries.

Acute abdominal condition. These products or other narcotics may obscure the diagnosis or clinical course of acute abdominal conditions.

Special risk patients. Administer with caution to certain patients such as elderly or debilitated patients and those with severe impairment of hepatic or renal function, hypothyroidism, Addison's disease, or prostatic hypertrophy or urethral stricture.

ADVERSE REACTIONS: Most frequently include lightheadedness, dizziness, sedation, nausea, and vomiting; more prominent in ambulatory than in nonambulatory patients; some may be alleviated if patient lies down; others include: euphoria, dysphoria, constipation and pruritus.

DRUG INTERACTIONS: CNS depressant effect may be additive with that of other CNS depressants. See Warnings.

For symptoms and treatment of overdosage and full prescribing information, see package insert.

Introducing EMPRACET® c̄ CODEINE #4

Each tablet contains: codeine phosphate, 60 mg (1 gr) (Warning—may be habit-forming); and acetaminophen, 300 mg.

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Our new non-aspirin/ codeine analgesic for moderate to severe pain.

New peach-colored Empracet c̄ Codeine #4 offers a potent alternative for patients in whom aspirin is not indicated.

Unlike compounds containing oxycodone which afford comparable analgesia, new Empracet c̄ Codeine #4 gives you CIII prescribing convenience—up to 5 refills in 6 months at your discretion (where state law permits). And, prescribing by telephone is permissible in most states. Moreover, new Empracet c̄ Codeine #4 has less addiction potential than does oxycodone.

For those of your patients requiring a less potent analgesic, non-aspirin Empracet® c̄ Codeine #3 provides effective relief of moderate pain.

Burroughs Wellcome Co. makes codeine combination products. You make the choice.

Wellcome

Burroughs Wellcome Co.
Research Triangle Park
North Carolina 27709

THE BOOK CORNER

New books received are acknowledged in this section and such acknowledgment must be regarded as sufficient return for the courtesy of the sender. Selection will be made for review in the interests of our readers and as space permits. Books are listed with advance data supplied by publishers. Prices quoted are not guaranteed. For further information, address queries to the publishers. Books here listed are available for lending from the Denver Medical Society Library.

Recent Acquisitions

ACKERMAN AND del REGATO'S CANCER: Juan A. del Regato and Harlan J. Spjut. 5th ed. St. Louis, Mosby, 1977. 1013 p. $51.50.
BASIC REHABILITATION TECHNIQUES: Robert D. Sine, ed. Germantown, Md., Aspen Systems, 1977. 228 p. $12.95. Gift.
BLOOD POLICY: Conference on Blood Policy, Washington, 1976. Washington, D.C., American Enterprise Institute, 1977. 212 p. Review.
BRAIN'S DISEASES OF THE NERVOUS SYSTEM: Walter R. Brain. 8th ed. New York, Oxford University Press, 1977. 1277 p. $29.50.
CLINICAL ONCOLOGY: John Horton and George J. Hill. Philadelphia, Saunders, 1977. 819 p. $30.00.
CORONARY ARTERY SURGERY: Thomas A. Preston. New York, Raven, 1977. 266 p. $12.50.
CURRENT SURGICAL DIAGNOSIS AND TREATMENT. J. Englebert Dunphy and Lawrence W. Way. Los Altos, Calif., Lange, 1977. 1139 p. $18.00.
THE DOCTORS AND PATIENTS HANDBOOK OF MEDICINES AND DRUGS: Peter Parish. New York, Knopf, 1977. 412 p. $5.95. Gift.
FEMALE PSYCHOLOGY: Harold P. Blum, ed. New York, International Universities Press, 1977. 454 p. Gift.
GASTROINTESTINAL BLEEDING: John A. Balint,. New York, Wiley, 1977. 101 p. $17.00.
GENETIC APPROACH TO HUMAN DISEASE.: Vincent M. Riccardi. New York, Oxford University Press, 1977. 273 p. Gift.
HANDBOOK OF OBSTETRICS AND GYNECOLOGY: Ralph C. Benson. Los Altos, Calif., Lange, 1977. 771 p. $9.50. Review.
HOSPITAL COSTS IN COLORADO: Patrick O'Donoghue. Denver, Spectrum, 1977. 148 p. Gift.
HOSPITAL REGULATION: American Hospital Association. Chicago, A.H.A., 1977. 188 p. $5.00.
INFECTIOUS DISEASES OF CHILDREN: Saul Krugman and others. 6th ed. St. Louis, Mosby, 1977. 539 p. $29.50.
INTRODUCTION TO ANESTHESIA: Robert D. Dripps. 5th ed. Philadelphia, Saunders, 1977. 557 p. $14.00.
MEDICAL ABBREVIATIONS AND ACRONYMS: Peter Roody. New York, McGraw-Hill, 1976. 255 p. $5.95.
MODERN CLINICAL PSYCHIATRY: Lawrence L. Kolb. 9th ed. Philadelphia, Saunders, 1977. 910 p. $17.50.
OCULAR THERAPEUTICS AND PHARMACOLOGY: Philip Ellis. 5th ed. St. Louis, Mosby, 1977. 283 p. $19.50. Gift.
PAGET's DISEASE OF BONE: Frederick R. Singer. New York, Plenum, 1977. 171 p. $19.50.
PATHOLOGY: W.A.D. Anderson. 7th ed. St. Louis, Mosby, 1977. 2 v. $37.50.
PURINE AND PYRIMIDINE METABOLISM: Symposium on Purine and Pyrimidine Metabolism, London, 1976. New York, Elsevier, 1977. 369 p. Gift.
RAGE, HATE, ASSAULT AND OTHER FORMS OF VIOLENCE: Denis J. Madden and John R. Lion, eds. New York, Spectrum, 1976. 265 p. $20.00.
RECONSTRUCTIVE PLASTIC SURGERY: John M. Converse,. 2nd ed. Philadelphia, Saunders, 1977. 7 v. $250.
REHABILITATION MEDICINE: Howard A. Rusk. St. Louis, Mosby, 1977. 675 p. $29.50.
RHOADS TEXTBOOK OF SURGERY: James D. Hardy, ed. 5th ed. Philadelphia, Lippincott, 1977. 2 v. $44.00.
SEPTIC SHOCK IN OBSTETRICS AND GYNECOLOGY: Denis Cavanaugh. Philadelphia, Saunders, 1977. 135 p.
STRESS TESTING: Myrvin Ellestad. Philadelphia, Davis, 1977. 296 p. Gift.
SYMPOSIUM ON RETINAL DISEASES: Symposium on Retinal Diseases, New Orleans, 1976. St. Louis, Mosby, 1976. 354 p. $37.50.
UROLOGIC PATHOLOGY: Myron Tannenbaum. Philadelphia, Lea & Febiger, 1977. 419 p.

Book Review

The Hospital at the End of the Santa Fe Trail: Clark Kimball and Marcus J. Smith, MD. Rydal Press, Santa Fe, New Mexico, 1977. 138 p. $8.95.

Until a history comes along that examines Santa Fe's handling of its medical problems from 1610 until 1865, when this brief history of St. Vincent's Hospital takes up events, this intriguing account will stand as an instructive and even inspiring lesson in establishing modern medical methods in a rude civilization.

At first it was the need and the sense of what must be done that turned wheels for the building of the hospital. As in matters of religion and culture, Jean Lamy, Bishop of Santa Fe, was the instrumental figure in the community's medical history. Care for the poor, the ill, and the young were paramount in his philosophy of action, and his resourcefulness was tried often.

The extensive introduction by Dr. Smith, this Journal's Scientific Editor for New Mexico, recounts many incidents of intrigue which advanced the Bishop's plans. On the unsettled frontier what could be predicted was that individuals would suffer or endeavor to surmount the cultural clash of settlers and Indians. This brought an ongoing tension.

A year or so after the Bishop arrived in Santa Fe he was joined by Father Etienne Avel, a young French priest. There had been ill will in Mora, a town over the mountains to the northeast, and in 1853 Father Etienne was sent there to replace the town's priest. According to the records, he arrived there unexpectedly to preside at mass. During the communion service, upon drinking the wine he realized he had been poisoned. Before his death he was able to direct that a hospital be established in Santa Fe with the $3,000 he would leave as his estate.

With this money the Bishop bought land near St. Francis, the old parish church, which contained tenements, and one of these old adobe buildings became in 1865 the first of its hospitals. It was under the direction of Sister Catherine of the Sisters of Charity, one of two strong Sisters who had come to work with the Bishop to expand services for the needy.

It was Vincent de Paul, canonized as Saint Vincent, who founded the Sisters of Charity in the 17th century for whom the hospital was named.

Poverty had been the established way of life at the Hospital until 1866 when the Legislative Assembly of the Territory of New Mexico provided an allotment of $100 each month, no grand largesse. The commissary privileges which were arranged for the Sisters by General of the Army James Carleton did greatly assist.

The Sanitorium at St. Vincent's Hospital in Santa Fe was completed in 1883, and until its destruction by fire in 1896 it was one of the town's most illustrious buildings with its iron mansard roof and lavish cupola.

By any measurement of hospital standards, St. Vincent's was substandard. It is said that Bishop Lamy would sit at supper under his umbrella whenever it rained, so leaky was the roof.

By 1876 St. Vincent's was operating under a policy of admitting all in need of medical attention, regardless of financial status, nationality, or religion. It helped that a physician and a pharmacist did not charge for their services. There was further support from General Carleton who gave $2,000 through the Soldier's Home Fund. The legislature gave $5,000, and area clergy provided $3,580.

The orphanage which was separate from the Hospital, and was a key element in the Bishop's plan, held 288 children; this number was in part large because of the unusually low death rate at St. Vincent's which maintained a rate of 14 per cent compared with the 50 per cent death rate that prevailed in national public hospitals.

Another memorable individual, Sister Blandina, came from Trinidad, Colorado, in 1877. She came to set up an industrial school for girls. Though trained in music, here she was establishing a brickyard - whose initial production of 250,000 bricks was a total disaster - from which ultimately solid bricks were to come for needed structures.

In 1880 plans were made for the Sanitorium, a three-story building which cost $25,000, complete with its steam-heat and ornate cupola. Following its opening in 1883, it rapidly became crowded with Easterners beset with tuberculosis, and was supplemented by another smaller building. The waves of the ailing subsided when Koch discovered the tuberculosis bacterium. In 1896 a fire destroyed the sanitorium

The effort to fund a new hospital was complicated by an oversight in which insurance on the sanitorium had not been paid up. Only $11,000 of the damage was covered. Santa Fe citizenry raised $10,000. In 1907 the legislature alloted $5,000 for a new building. The Sisters of Charity borrowed $25,000 at 4 per cent interest, and by 1910 a $75,000 building was completed.

The town grew and with it hospital needs accumulated. The growth in the 1930's and 1940's was heavy. In 1951 work started on a new hospital on East Palace Avenue which was completed in 1953. Because of the continuing policy of free admission to indigents a large indebtedness built up From 1955 to 1965 this was $1.7 million. A referendum or a tax levy to aid the hospital was defeated, so other efforts to reduce the indebtedness were made, with enough success, along with the arrival of Medicaid and Medicare, so that by 1967 the deficit was down to $275,000, at which time the legislature was in a mood to adopt a .25 per cent sales tax to aid indigents at the hospital.

Again, in 1975 St. Vincent's required expanded facilities, and a purchase of 40 acres on St. Michael's Drive outside the old town has placed St. Vincent's in a new era, where assuredly those traditions which infused this institution with such pride will continue to prevail.

Allen Young

A Man Possessed: The Case History of Sigmund Freud: Nat Morris. Los Angeles, Regent House, 1974. 160 p. Cloth $7.95, Paper $2.45.

The tiny advertisement of this book in the November, 1975 *Atlantic Monthly* quoted Walter C. Alvarez, MD, " . . . the best study of Freud I have ever read explaining the events responsible for his ideas. . .', and H.J. Eysenc, University of London ". . .an unusual and intriguing book. . . the writer has many important and interesting things to say."

The jacket expands these quotations, and quotes C.G. Jung, he was a man possessed by a demon—a man vouchsafed an overwhelming revelation that took possession of his soul and never let him go."

Nat Morris' scientific education was obtained as a salesman for medical publishing concerns, which also ensured a wide acquaintance among physicians. His book, THE CANCER BLACKOUT, as Maurice Natenberg, Regent House, Chicago, 1959 packs a real clout. He came to know and admire Dr. Coyne H. Campbell, whose *The i'sychopathy of Freudianism* struck a responsive chord.

Morris has read almost everything by or about Freud, and this small book of 160 pages places—in the opinion of this reviewer—Sigmund Freud exactly in his correct niche.

Horace E. Campbell, M.D.

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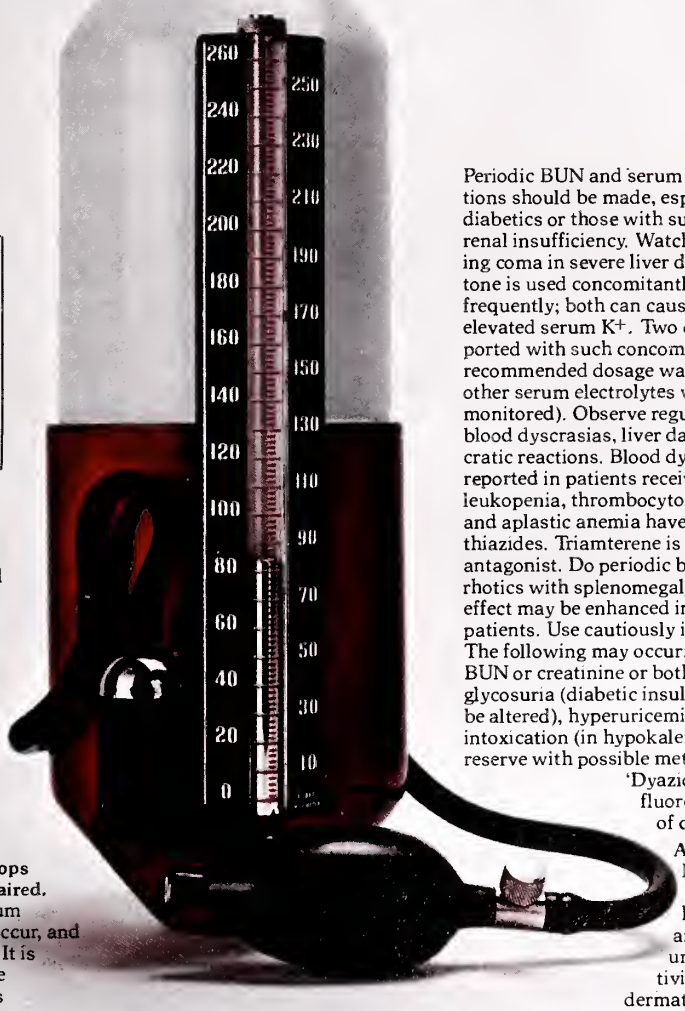

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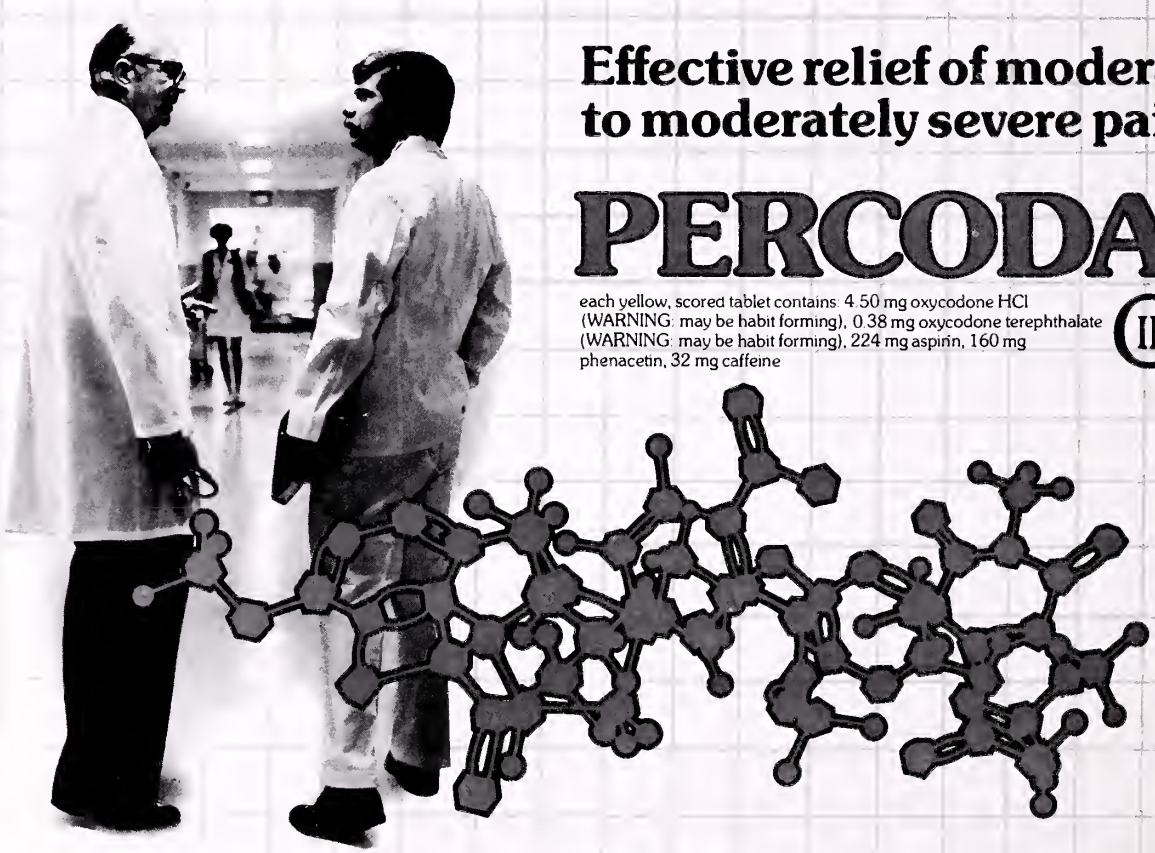

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Please see facing page for Brief Summary of prescribing information.

Tablets

Percodan® Ⓒ II

DESCRIPTION Each yellow, scored tablet contains 4.50 mg. oxycodone HCl (WARNING: May be habit forming), 0.38 mg. oxycodone terephthalate (WARNING: May be habit forming), 224 mg. aspirin, 160 mg. phenacetin, and 32 mg. caffeine.

INDICATIONS For the relief of moderate to moderately severe pain.

CONTRAINDICATIONS Hypersensitivity to oxycodone, aspirin, phenacetin or caffeine.

WARNINGS Drug Dependence Oxycodone can produce drug dependence of the morphine type and, therefore, has the potential for being abused. Psychic dependence, physical dependence and tolerance may develop upon repeated administration of PERCODAN®, and it should be prescribed and administered with the same degree of caution appropriate to the use of other oral narcotic-containing medications. Like other narcotic-containing medications, PERCODAN® is subject to the Federal Controlled Substances Act.

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DOSAGE AND ADMINISTRATION Dosage should be adjusted according to the severity of the pain and the response of the patient. The usual adult dose is one tablet every 6 hours as needed for pain.

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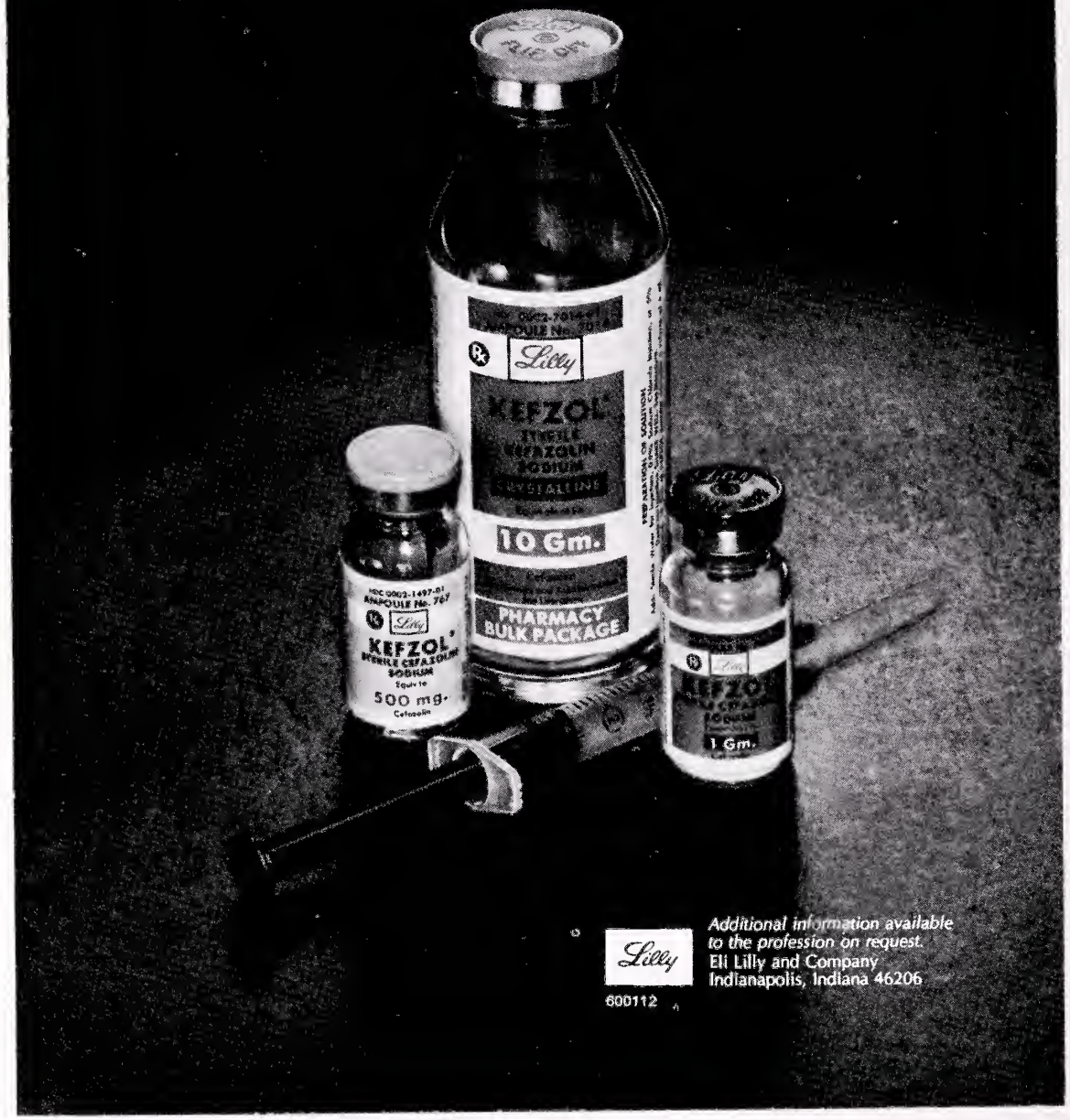

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A psychiatrist, a friend of mine, asserts as a basic truth that a little paranoia serves a protective role in an integrated personality. So if the examples I bring to you today seem to represent threatening social and economic pressures bearing upon your practice, do not feel insecure about your emotional balance, consider instead my presentation as simply illustrative of a few social forces affecting your relationship with your patient. You will notice I did not use the phrase "Delivery of Health Care" as I believe this sounds too much like carrying groceries. Perhaps, in fact, this is one of the goals of modern social planning, to reduce the more skilled professions to a common level by constantly applying to that skill a commodity like designation rather than a title, suggesting respect.

Social Forces on the Doctor-Patient Relationship

Where better to start than at the beginning with admission to Medical School. The capitation system now encourages the selection of designated minority students at the expense of better qualified non-minority candidates. I would remind you that the supreme court has agreed to hear the case of a Board of Regents of the University of California vs a University of California Davis Student (the Bakke case) who won his case in the California courts seeking admission to Davis on the grounds that he was passed over in Medical School selection and less qualified minority students were taken instead. If the Regents of California lose in the supreme court test, it may signal the end of reverse discrimination in Medical School selection systems. I refer you also to the recent editorial in the March 1977 New Mexico State Society Newsletter by Doctor R. C. Derbyshire alluding to the quota system for the training of specialists, this to be accomplished by the Federal Government dictating the numbers of trainees who can be taken into residency programs.

Address to the American College of Surgeons New Mexico Chapter American College of Surgeons, May, 1977.

Moving to the actual day-to-day care we provide. We now hear that a mass of unnecessary surgery is being done in the United States. It is implied, in fact, that any surgery which is elective may possibly be unnecessary. The proposed solution is to force upon us through third party providers, the acquisition of a second opinion for all scheduled elective surgery. The immediate effect would be a potentially staggering added cost. Of course the argument is advanced that if one three-hundred dollar operation could be cancelled through second opinion screening, this would pay for six fifty dollar consultations that were not needed, and the program would still be even on a financial basis. I don't need to remind you that just around the corner is a plantiff's attorney waiting for that consultant who said the operation was not needed, only to have something happen to the patient conceivably attributed to the withholding of surgery—let alone the almost impossible dilemma of deciding what is that fine line between absolute necessity and that which is preferable for health reasons. This has moved the American College of Surgeons to affirm the time honored position that every patient has the right to another opinion but that such should not be required.

There are few areas which affect the surgeon more than his continued relationship with the hospital. The more complex our skills become the more we will become intertwined with the hospitals in the care of patients. I cite a few of these interrelations most pressing at this moment. Equipment availability is now coming under the Health Planning Act in most states. In our last legislative session a bill was presented and defeated which would require the Health Planning Council to approve anything costing more than $100,000. I urge each of you to look at Public Law 93691 which, if allowed to stand, will mandate the Health Planning Council control of hospitals and medical practice by 1980. Fortunately this bill is currently being challenged in the courts by the State of North

Carolina and the North Carolina Medical Association. At this time the bill is law and it provides all the mechanisms necessary to completely federalize medical practice.

As hospital services have increased in complexity, hospital budgets have risen astronomically. Thus, if you consider hospitals as an industry along with General Motors, Ford, US Steel, etc., hospital budgets rank fifth in the total number of dollars they put into circulation in the form of salaries paid and services purchased. In New Mexico they rank second only to the Federal Government in the amount of money placed into circulation in this state. As hospitals grow, federal requirements are imposed upon them, if they are to participate in federal programs. There is no money free of strings you know. And as hospitals are dictated to, so they tend to become subtly more dictatorial. Not dictatorial in the sense of a Monarchy, that would offend our democratic principles. Rather their dictatorship assumes the honored bureaucratic gambit of control through the rule-making power of regulatory agencies. Thus the development of peer review both preadmission and during the hospital stay, the gradual increase in the number of full time employed physicians in hospitals, the hint that we will soon need a full time employed chief of each hospital department, and most recently the attempt to specify in detailed form the surgical privileges one may exercise. It is no longer possible to apply for privileges in General Surgery and have any administrator admit what this means. At the moment this is true mainly in larger hospitals. How soon it will reach into hospitals of all sizes I cannot predict, but I would like to bring to your attention a set of figures that may surprise you.

The concerns of small hospitals have long been a challenging problem for the American College. In 1957 the Board of Governors at its annual meeting began discussing surgery in small hospitals. There are now 5942 hospitals registered and approved in the United States by the American Hospital Association. Of these 3159 or 53 per cent are less than 100 beds, 1292 or 22 per cent are one to two-hundred beds, and 1491 or 25 per cent are over 200 beds. In New Mexico 38 hospitals, or 72 per cent are less than 100 beds, 9, or 17 per cent, are 100 to 200 beds and only six, or 11 per cent, are over 200 beds. Thus 85 per cent of our hospital beds in this state are in hospitals of less than 200 beds. On a national basis taken from the PAS System and the CPHA (Commission of Professional and Hospital Activities) 12 per cent of operations are done in hospitals of less than 100 beds and 31 per cent of operations are done in hospitals of less than 200 beds.

Today the larger hospitals tend to have more highly trained men, but it has long been the position of the Board of Regents that since so much surgery is done in small hospitals the training of surgeons must continue to be broadly based in general surgery, if the surgeon is to have the requisite skills to care for patients presenting in small hospitals. In their 1974 report to the Board of Governors, the Committee on the study of care in small hospitals concluded that the hospital accreditation does not adequately survey operative skills and operative care in these hospitals and that hospital boards do not automatically develop hospitals which are appropriate to the local community or the need of the surgical patients.

Recent pressure upon the surgeon-patient relationship could not be more acute than in the total area of malpractice. You are aware of our newly formed insurance company in New Mexico. I am struck by the enormous potential power of this company. In their underwriting policies they can deny a community its physician. The Underwriting Committee obviously could refuse to accept any physician but that one most clearly demonstrated to be risk free, and once a claim has been brought against that physician, simply cancel his policy. The company would thus have the greatest possibility of financial success as an insurance company, but it would also be serving the public rather poorly. In our legislative efforts two sessions ago we failed in our effort to have included in our law a control of contingency fees, a locality rule for witnesses, a revelation of colateral sources of income when trying a malpractice case and the detailing of what would be necessary to prove a lack of informed consent. It is possible that our law only allowed time for the formation of a new company. I believe it is too early to know whether this company can succeed without further tort reform, but certainly none of us is unaware of the influence of the threat of negligence upon our practice.

It is not possible to leave this subject without comment upon continuing medical education, improved skills and re-licensure, and recertification examinations as proof of compe-

tence. I wish it were possible to assume that expertise in medical practice was an assurance of freedom from malpractice action. This sadly is not the case—Goat Gland Brinkley never had a suit against him. But seriously the study of the National Association of Insurance Commissioners would indicate that the more highly skilled a physician, the more likely he is to be sued. Apparently, the more complex the case you perform, the more chance there will be some sort of bad result or dissatisfying result to the patient, and in the present climate, negligence or not, a dissatisfied patient is enough to generate a law suit. There is at least one New York attorney writing in National Review a year ago who advocated the solution to the problem as a change in statute to require that negligence shall be proved beyond a reasonable doubt before it can stand as a conviction. At present in contrast all that is required is that one physician witness testify to an action being negligent and the patient has a case.

Finally, if that were not enough I would remind you of the incredible number of dollars of our tax money being yielded by the various federal bureaus in their entry into health care. The most recent to come to my attention is the possibility of obtaining a grant from NIH for an effort to discover hypertension and lung disease in employee groups. Applications have been invited for the securing of the grants from NIH. It makes no difference apparently that the group of employees may already have a medical program in which they are either having periodic or pre-employment physicals. Rather it is money available as far as I can determine to re-duplicate work and assemble information already available. But of course it is a bureau perpetuated by dreaming up actions requiring money expenditure whether the expenditure accomplishes anything or not. If the budgets show that the money was spent it is likely that the money will be re-appropriated from tax funds again next year. All of which can only lead one to conclude that the age old observation is correct; when the faucet is stuck wide open it is easier to reach for a "tin cup" to put under it than to look for a pipewrench to fix it.

W. W. Kridelbaugh
Albuquerque, New Mexico

Suicide In Western States

In 1970 the average suicide rate for the United States was 11.6 per 100,000 population. For the years 1969-1971, the rates for the eleven continental Western States exceeded the national average in all but one state, and were twice as high in three states, according to a recently published article emanating from the Los Angeles Suicide Prevention Center.[1] The age standardized rates are as follows:

State	Standardized Rate per 100,000 population
Nevada	28.78
Arizona	22.40
Wyoming	20.53
Colorado	17.98
California	17.27
New Mexico	16.16
Idaho	15.87
Oregon	15.23
Washington	13.26
Montana	12.19
Utah	11.39

By commiting suicide one rejects the community in which one lives: the victim believes that society has failed to make life worthwhile. Thus the suicide rate is a measure of the mental health and social deviance of a community. To gain insight into suicide, one must probe into demographic and social integration factors such as age, sex, educational level, population density, ethnic composition, crime and divorce rates, transiency, family characteristics, and poverty. These community variables influence the suicide rate.

Other important variables which affect the suicide rate are the backgrounds of coroners, the methods they use to identify suicides, and the extent of professionalism in their staffs. Are suicides properly certified?

The investigators sampled population and coroners' offices in 191 of 411 counties in the Western States. Data were collected from large (more than 30,000 population) and small (less than 30,000 population) counties.

Populations were about even in sex distribution. Whites averaged 77,000, non-whites, 4,000. Twelve per cent were foreign born. The

population had increased 11 per cent in the previous ten years. The educational level was 12.4 years. Sixty-five per cent of the counties were rural.

The unemployment rate was 6 per cent. The poverty rate was 18 per cent. Twenty-three per cent of the employed were in professional or managerial positions. Fifty-five per cent lived in houses built prior to 1950. Ten per cent of the population lived in crowded areas. Eighty-eight per cent owned one or more automobiles. Money spent by government agencies for health and welfare averaged $312 per capita per year. Sixty-two per cent voted in the 1968 presidential election.

The above description is representative of the Western States, and indicates that the counties tended toward the high socioeconomic levels.

Coroners had diverse backgrounds: physicians, 32 per cent; morticians, 23 per cent (all coroners in Wyoming are morticians); police officers, 16 per cent; as well as lawyers, public administrators, and justices of the peace. Twenty-five per cent had only high school education.

Fifteen per cent of the coroners were medical examiners. In Oregon and New Mexico, appointed medical examiners are responsible for statewide services—the University of New Mexico School of Medicine houses the coroner's office. In Colorado, an administrator of health and hospitals also serves as coroner of Denver. In Wyoming, Idaho, and Montana, coroners utilize inquests and coroners' juries. In these states coroners have considerable independence, too few pathologists, and inadequate funding.

In order to certify a death as suicide, some coroners require a note, seventy-six per cent believe in obtaining autopsy data in cases of homicide, but only 30 per cent in suspected suicide, and 13 per cent in apparent accidents.

It has been said that family physicians and family members tend toward failing to report suicide because of a societal taboo. Suicide can be an inconvenience and embarassment to survivors. Are coroners immune? The study shows that 37 per cent of coroners try to avoid the label of suicide in order to maintain the reputation of the deceased and the family.

Previous reports indicate that in doubtful situations physician coroners are less rejecting of suicide as a cause of death than are coroners with different backgrounds. The present study indicates that pathologists and law officers were non-rejecting, other physicians were at or below the mean level of the suicide rate, and lawyers diagnosed suicide the least—perhaps because they are hesitant to accept less than complete proof of the cause of death.

To make a diagnosis of suicide, coroners consider the following factors in combination: personal history of the victim, 79 per cent; suicide note, 65 per cent; history of previous attempt, 49 per cent; shot within arm length, 47 per cent; and location of wound, 50 per cent.

There are coroners who tend to reject suicide as the cause of death, and others who do not. The analysis of coroners' offices' characteristics indicate that 40 of 16 variables accounted for 14 per cent of variability in the suicide rates. Coroners who stress loss of a loved one as a criterion for establishing intent, who order a large number of autopsies, and who give credence to statements of others, more often decide that suicide was the means of death, and hence account for higher suicide rates than elsewhere.

In large counties, coroners most predictive of high suicide rates were more accepting of a diagnosis of suicide, more concerned with the personal aspects of the deceased and with the physical circumstances of the death.

In small counties, because of limited personnel and facilities, coroners concentrated on the physical aspects of the death rather than the personal, and by implication were more rejecting of suicide than were their peers in the large counties.

Suicide rates differ in large and small counties. In large counties where families possess one or more automobiles, more often live in new houses, and with a large per cent of the work force in professional or management positions, the suicide rates were higher than in counties which did not show these features. Among the small counties, suicides were greatest in the wealthiest rather than the poorest.

Coroner office variables in large counties explain 37 per cent of variations in reported suicide rates, whereas community variables account for 41 per cent. In small counties the variations were less, 24 and 13 per cent respectively.

A high suicide rate is consistent with a highly mobile and transient population as found in the West, where 55 per cent of the population have moved into homes within five years prior to the

years of the study. On the other hand, a population with a preponderance of young people would be consonant with a low suicide rate because the greatest incidence of suicide is among the aged and middle-aged—yet in seven Western States the age distribution is skewed toward the young.

Unmentioned in the article are other influences which might affect the suicide rate. One is the weather. The sun-drenched land, the big sky, and the energizing air characterize the West. For more than a century refugees from the miasmas of the Mississippi Basin and from the smoggy industrialized Northeast have migrated hopefully to the West, searching for physical renewal. Curiously, several major statistical analyses[2] indicate that most suicides occur in the spring and summer (other reports differ), suggesting that a high suicide rate is appropriate for parts of the country where most of the year the weather is summery and springy.

From another vantage point, suicide rates of 19.9 or more are rare. In international tabulations only Hungary, Austria, West Germany, Czechoslovakia, Sweden, and Finland, have rates as high as Nevada, Arizona, and Wyoming. Obviously these are disparate geographic and ethnic entities. Perhaps a perceptive reader can explain the similarity.

In addition to increasing the mystique surrounding suicide, the study has uncovered a need for improvements in the coroner system, as suggested by the coroners themselves: death reports, certificates, and procedures need updating; funds are required for additional autopsies; greater professionalism is essential; formal requirements for training of coroners; increased quality control over staff performance; and the need for services in small counties.

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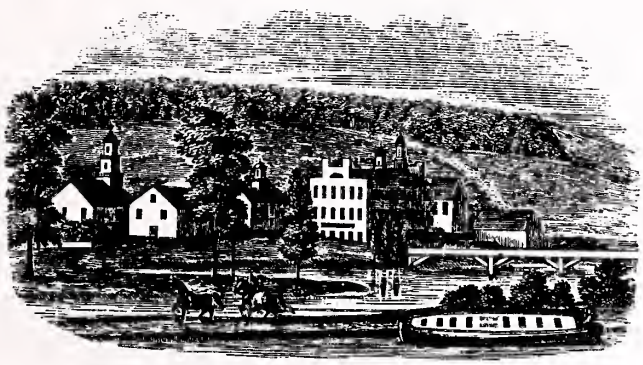

Diabetic ketoacidosis

Counter-Regulatory Hormones Versus Insulin in Therapeutic Response

Report of a Case

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Counter-regulatory 'stress' hormones, namely, glucagon, growth hormone, catecholamines, and cortisol have been observed to be elevated in patients with diabetic ketoacidosis.[1] While much evidence has been published supporting a role for these counter-regulatory hormones in the development of ketoacidosis, it is not known whether or not normalization of the plasma concentration of these hormones is necessary to correct the metabolic derangement. In the present case report we have examined the acute simultaneous response of three counter-regulatory hormones to the rise in circulating plasma free insulin concentration following administration of exogenous insulin. Our studies confirm the reported elevation of these ketogenic hormones in diabetic ketoacidosis and demonstrate the reduction in glucagon concentration with insulin replacement, the failure of growth hormone levels to closely relate to insulin replacement, and an unreported reciprocal response in plasma cortisol concentration relative to simulataneous insulin levels. In this patient, normalization of glucagon, but not of cortisol or growth hormone, was closely related to clinical correction of diabetic ketoacids.

CASE REPORT

A 20-year-old causasian male presented with stupor and hyperventilation. He was known to have insulin dependent diabetes mellitus for eight years treated with 80 units of NPH U100 insulin daily, and a history of multiple previous episodes of diabetic ketoacidosis.

Physical examination on admission showed a non-obsese, semi-comatose white male who responded only to painful stimuli. Vital signs included weight of 71 kg, height 73 inches, respirations 36/minute, sinus tachycardia of 132 beats/minute, blood pressure 110/65, and rectal temperature 38°C. A right posterior auricular abscess was present. Deep tendon reflexes were depressed. Chest x-ray was interpreted as demonstrating a hazy infiltrate in the middle lobe of the right lung. A urine toxicology screen showed only the presence of ketone bodies.

Initial laboratory data included a plasma glucose concentration of 690 mg/dl, plasma ketone bodies positive at four dilutions (Acetest Tablets*), arterial pH of 6.80, plasma bicarbonate concentration of 3.0 mEq/L, and a BUN of 26 mEq/L. Initial therapy included rehydration with 0.15 M sodium chloride, administration of alkali in the form of sodium bicarbonate, and intravenous penicillin G, 250,000 units.

Following two basal blood samples, insulin therapy was begun with seven units of regular insulin intramuscularly and repeated at hourly intervals.[4] During the first two and one-half hours of therapy, the patient's condition did not improve and his hyperglucemia and ketoacidosis persisted (Fig. 1). At this point, high dose intravenous insulin treatment was initiated with a 50 unit "bolus" of U100 regular insulin plus a constant infusion of 100 units per hour intervenously utilizing a Harvard infusion pump.[5] This regimen resulted in an abrupt reduction in plasma glucose concentration to 423 mg/dl with a concommitant increase in arterial plasma pH and bicarbonate concentration. Following two hours of high dose dose insulin therapy, the insulin dosage was reduced to an infusion of 10 units per hour and eventually 1.0 units per hour during the last two hours of the observation period. With correction of plasma chemistries within 18 hours of admission, mental status improved and he was discharged one week post admission on 80 units of NPH insulin daily.

Methods: Plasma electrolytes were measured with a Technicon SMA 6/60 Analyzer, Technicon Instrument Company, Tarrytown, New York. Blood gases and pH were analyzed by a PHM MK2 Digital acid-base Analyzer, Radiometer Corp., Copenhagen, Denmark. Plasma betahydroxybutyrate and acetoacetate were assayed within 12 hours of collection by enzymatic analysis. Plasma free fatty acids were measured by the method of Dole with the modification of Duncombe. Utilizing this methodology, plasma was extracted with a heptane-alcohol-water mixture and corrected for the contribution of ketone bodies. Plasma insulin, glucagon, growth hormone, and cortisol were assayed by standard radioimmunoassay techniques. Prior to insulin assay, endogenous insulin antibodies were precipitated with 25% polyethylene glycol.

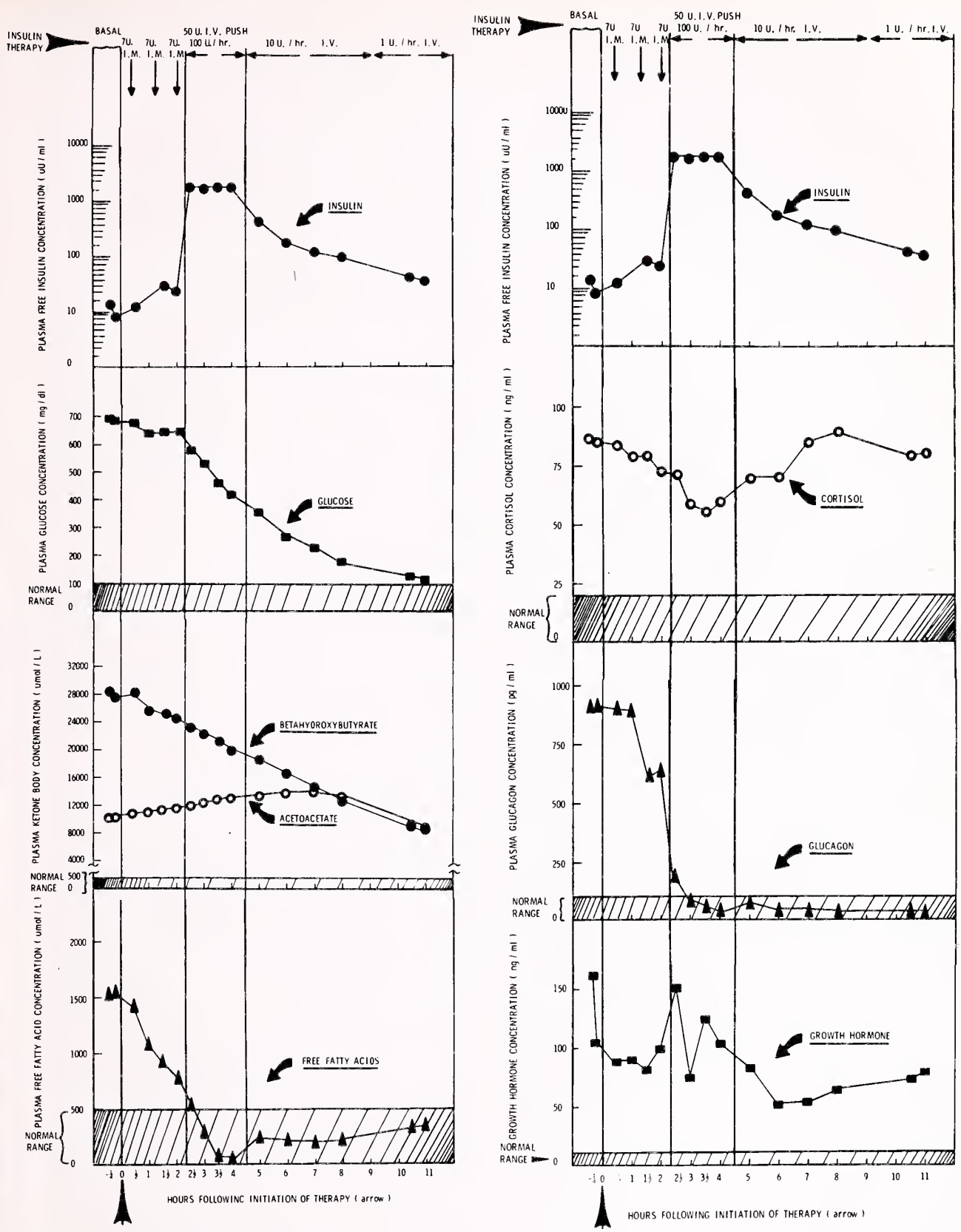
TEMPORAL RELATIONSHIP BETWEEN PLASMA INSULIN AND HORMONALLY MODULATED SUBSTRATES IN KETOACIDOSIS
TEMPORAL RELATIONSHIP BETWEEN PLASMA INSULIN AND COUNTER - REGULATORY HORMONES IN KETOACIDOSIS
INSULIN THERAPY
BASAL
7U. I.M.
7U. I.M.
7U. I.M.
50 U. I.V. PUSH
100 U. / hr.
10 U. / hr. I.V.
1 U. / hr. I.V.
PLASMA FREE INSULIN CONCENTRATION (uU / ml)
INSULIN
PLASMA GLUCOSE CONCENTRATION (mg / dl)
GLUCOSE
NORMAL RANGE
PLASMA KETONE BODY CONCENTRATION (umol / L)
BETAHYDROXYBUTYRATE
ACETOACETATE
PLASMA FREE FATTY ACID CONCENTRATION (umol / L)
FREE FATTY ACIDS
HOURS FOLLOWING INITIATION OF THERAPY (arrow)
PLASMA CORTISOL CONCENTRATION (ng / ml)
CORTISOL
PLASMA GLUCAGON CONCENTRATION (pg / ml)
GLUCAGON
GROWTH HORMONE CONCENTRATION (ng / ml)
GROWTH HORMONE

Fig. 1. Temporal relationship between changes in plasma insulin concentration (top panel) and the simultaneous changes in counter-regulatory hormones (left) and hormonally sensitive substrates (right) in diabetic ketoacidosis (lower three panels). Observations were during the therapeutic administration of exogenous insulin as indicated.

Plasma Free Insulin Concentration: Pretreatment plasma free insulin concentration on two separate samples was 14 and 8μ U/ml respectively as shown in Fig. 1. In spite of the fact that a total of 21 units of intramuscular insulin was administered within a two hour period, plasma free insulin concentration did not exceed 36μ U/ml when assayed at 30 minute intervals during this period. Ten minutes following the bolus injection of insulin, a concentration of 1,621μ U/ml was present which was sustained during the constant infusion of 100 Uhr. Reducing the insulin infusion to 10 U/hr, resulted in a plasma free insulin concentration of 117μ U/ml. Further reduction in the infusion rate to 1.0 U/hr. resulted in a plasma insulin concentration . of 48μ U/ml.

Plasma Glucagon Concentration: Pretreatment plasma glucagon concentrations were 900 pg/ml and 916 pg/ml respectively on the two baseline blood samples obtained prior to initiating therapy (normal less than 100 pg/ml). A rapid decline in glucagon concentration to 30 pg/ml occurred during intravenous insulin infusion of 100μ U/ml. Plasma glucagon then remained within our physiologic range for the duration of the observation period.

Plasma Cortisol Concentration: The two initial blood samples drawn prior to initiating therapy contained cortisol concentrations of 86μ g/dl and 84μ g/dl respectively (normal less than 20μ g/dl), and they remained elevated throughout our observation. During the period of pharmacologic insulin concentrations of greater than 1,000μ U/ml, the nadir of 54 g/dl of cortisol concentration was observed and with the return of plasma free insulin to physiologic concentrations, a progressive rise in plasma cortisol concentration to 92μ g/dl occurred. This reciprocol relationship to the change in plasma insulin concentration is graphically shown in Fig. 1.

Plasma Growth Hormone: Plasma growth hormone concentration was 165 ng/ml and 105 ng/ml (normal less than 10 ng/ml) on the two basal blood specimens drawn prior to initiating therapy. During the infusion of pharmacologic dosages of insulin, much variability in growth hormone concentration was observed as shown in Fig. 1. Although growth hormone concentration gradually attained a nadir of 52 ng/ml toward the end of the study when plasma insulin concentrations were within the physiologic range, growth hormone never obtained normal concentrations.

Plasma Glucose Concentration: Pretreatment plasma glucose on two separate samples was 690 and 694 mg/dl respectively. This concentration demonstrated an initial small decline during the intramuscular injection of 7 U/hr with a consequent elevation of plasma insulin of 36μ U/ml. Ten minutes following the elevation of plasma insulin to levels exceeding 1,000μ U/ml, a rapid decline in glucose concentration was observed of approximately 115 mg/dl/hr (Fig. 1). This rapid decline continued until plasma insulin levels were reduced toward 100μ U/ml when the rate of decline in plasma glucose fell to 60 mg/dl/hr. The reduction in the concentration of glucose coincided with the reduction in the concentration of glucagon as well as with the elevation in plasma insulin. In contrast, no direct relationship between the decline in glucose concentration and the alteration in cortisol or growth hormone was evident.

Plasma Betahydroxybutyrate: Basal plasma betahydroxybutyrate concentration was 27,906μ mol/L and 28,086μ mol/L respectively in the two initial blood samples. During treatment with insulin, a fixed rate of decline of betahydroxybutyrate was observed independent of changes in insulin concentration. Thus, the betahydroxybutyrate disappearance rate of 2,216μ mo/L/hr observed during plasma insulin concentrations in excess of 1,000μ U/ml was similar to the disappearance rate of approximately 1,916μ mol/L/hr during the final period of insulin therapy with insulin levels of less than 100μ U/ml. Of the three counter-regulatory hormones assayed, only the reduction in plasma glucagon temporally correlated with the simultaneous reduction in the concentration of this substrate.

Plasma Acetoacetate: Basal plasma acetoacetate concentration was 10,082μ mol/L and 10,185μ mol/L on the two blood samples obtained prior to therapy. With initiation of insulin therapy, a progressive increase in plasma acetoacetate concentration was observed. During the latter half of the observation period when plasma insulin concentrations were in the physiological range of 100μ U/ml, a decline in acetoacetate concentration occurred.

Plasma Free Fatty Acids: Pretreatment plasma free fatty acids (FFA) were elevated on admission to 1,532μ mol/L and 1,545μ mol/L in the two initial samples. With initiation of intramuscular insulin therapy and attainment of plasma insulin concentrations of only 50μ U/ml, an immediate and rapid decline of approximately 412μ mol/L/hr in plasma free fatty acid concentration occurred. This rate of decline of approximately 383μ mol/L/hr continued throughout the administration of pharmacologic dosages of insulin. During the final period of observation when circulating plasma concentration of free insulin was approximately 100μ U/ml, plasma free fatty acid concentration rose but remained within normal range. In addition, the decline in FFA concentration paralleled the reduction in plasma glucagon concentration (Fig. 1).

Plasma Bicarbonate and Arterial pH: Plasma bicarbonate concentration was 3.0 mEq/L on both basal blood specimens (arterial pH = 6.8). This concentration remained unchanged until pharmacologic doses of insulin were administered which initiated a progressive increase in both bicarbonate concentration and arterial pH so that at the end of the eleven hour observation period arterial pH was 7.23 and plasma bicarbonate was 11.0 mEq/L.

Discussion

At least four counter-regulatory hormones may contribute to the development and/or maintenance of diabetic ketoacidosis by enhancing lipolysis and/or hepatic ketogenesis. The concentration of each of these four counter-regulatory hormones has been reported to be proportional to the severity of diabetic ketoacidosis and with therapy, these hormones eventually return to basal concentration. However, which specific hormone(s) must be normalized with insulin administration to correct hyperglycemia and ketosis, is not resolved. Nor is the specific concentration of plasma free insulin required for counter-regulatory hormone normalization defined.

Our study extends previous reports of the therapeutic response to insulin in diabetic ketoacidosis[1, 4, 6-8] by relating the plasma free insulin concentration to the simultaneous rate of

change of both hormonally sensitive substrates and counter-regulatory hormones. Our results suggest that plasma glucagon, but not plasma cortisol or growth hormone, is rapidly reduced in concentration with insulin therapy. Finally, our data indicate the value to the clinician of assaying plasma insulin levels utilizing polethylene glycol precipitation of endogenous antibody, to monitor the plasma insulin response to various routes and dosages of hormone therapy. As a preliminary guideline to dosage, a plasma insulin level of 1,000u U/ml would appear to achieve immediate normalization of plasma glucagon concentration, inhibition of FFA production, and initiation or reduction in plasma ketones and glucose. Sustained levels of 100u U/ml may be considered appropriate thereafter providing continued suppression of plasma glucagon, and reduction in glucose and betahydroxybutyrate.

Summary

The contribution of counter-regulatory hormones to the response to treatment of diabetic ketoacidosis is unresolved. This case report demonstrates the temporal changes of plasma cortisol, glucagon, and growth hormone in relation to the therapeutic reduction in plasma glucose, free fatty acids, and ketones, during insulin therapy of ketoacidosis. We observed that sustained plasma insulin levels of 100u U/ml were sufficient to maintain suppression of glucagon concentration and to sustain the progressive correction of hyperglycemia and hyperketonemia. Our experience suggests that in this patient, glucagon, but not growth hormone or cortisol, is a counter-regulatory hormone which may require therapeutic reduction in order to satisfactorily correct diabetic ketoacidosis.●

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Cardiac disease and recurrent gastrointestinal bleeding

Angiodysplasia with accompanying hypertrophic subaortic stenosis

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The association of chronic lower gastrointestinal bleeding with angiodysplasia and aortic valvular stenosis has been recognized for some time. Less commonly recognized, however, is a similar association with idiopathic hypertrophic subaortic stenosis (IHSS). We are reporting such a case proven by cardiac catheterization and visceral angiography. The combination of recurrent, undiagnosed lower gastrointestinal bleeding in an elderly patient with left ventricular outflow obstruction should prompt vigorous and thorough angiographic search for angiodysplasia of the bowel.

CASE REPORT

A 57-year-old white female was referred for evaluation of recurrent, painless gastrointestinal bleeding. An extensive workup prior to her referral had included barium examination of the stomach, small bowel, and colon, as well as proctoscopy and visceral angiography. Superficial gastritis was diagnosed at endoscopy, and a vagotomy and antral resection were performed. However, gastrointestinal bleeding recurred. A six year history of decreasing exercise tolerance, angina, and shortness of breath had been accompanied by a cardiac murmur.

On admission to Bernalillo County Medical Center, examination showed a moderately obese woman with blood pressure of 100/60 with regular pulse of 88. Peripheral pulses were symmetrical. A brisk carotid upstroke with a questionable bifid pulse was discovered. The precordium was quiet with a diffuse PMI. A grade 2/6 crescendo, decrescendo systolic murmur was heard best between the apex and lower left sternal border. The murmur increased with the Valsalva maneuver and in the upright position, but decreased in the squatting position.

Admission hematocrit was 24 per cent. Electrocardiogram demonstrated marked left ventricular hypertrophy with normal axis. Echocardiogram demonstrated systolic anterior motion of the mitral valve with asymmetric septal hypertrophy. The septum measured 15 mm. in thickness. Chest x-ray demonstrated slight enlargement of the left ventricle and normal pulmonary vasculature.

A cardiac catheterization (Table I) demonstrated normal right heart pressures and a resting gradient of 50 mm. mercury that was abolished by intravenous propanolol. A bi-ventricular cineangiogram demonstrated a thickened inter-ventricular septum with the ratio of upper to lower septae being 0.6. The coronary arteries were angiographically normal.

During her course in the hospital, the patient experienced continued angina along with one episode of painless lower gastrointestinal bleeding. A selective superior mesenteric arteriogram was performed. No evidence of active bleeding was demonstrated, however a small dense reticular collection of dilated arteries was noted along the antimesenteric border of the ascending colon, 6 cm. above the level of the ileocecal valve. Early venous filling of vessels supplying this particular site of the colon was noted (Figs. 1 a & b). No other such lesions of the gastrointestinal tract were demonstrated on selective inferior mesenteric and celiac arteriograms. These findings were consistent with angiodysplasia.

Gastrointestinal bleeding recurred and a right hemicolectomy was performed. On pathologic examination of the surgical specimen, purplish red discolorations of the mucosa were observed at multiple levels. Histologic sections in the region of the angiographically demonstrated lesion showed mucosal thinning with large, distended submucosal capillaries. Deep to this were clusters of large thin walled veins, several of which suggested previous thromboses (Fig. 2).

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Fig. 1 a. Superior mesenteric arteriogram. Mid arterial phase. Opacification of the superior mesenteric artery demonstrates early filling of a small dense localized collection of ectatic vessels along the antimesenteric border of the ascending colon.

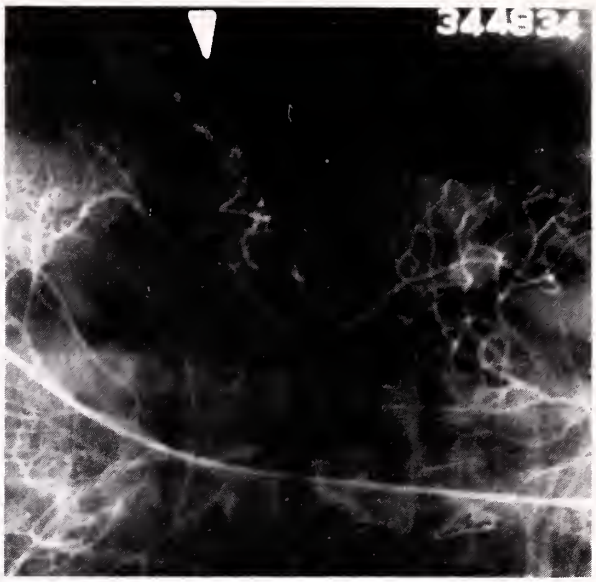

Fig. 1 b. Superior mesenteric arteriogram. Late arterial phase. Early filling of a large vein is demonstrated and is compatible with an arteriovenous malformation.

Discussion

Angiodysplasia was first described in 1949 by Gentry as an arteriovenous malformation most commonly affecting the stomach.[1] Numerous investigators have confirmed this finding, but have identified the cecum and right colon as the most common location. The association of gastrointestinal bleeding with aortic stenosis was suggested in correspondence to the *New England Journal of Medicine* by Heyde and Schwartz in 1958,[2,5] and in a later study in 1961 by Williams who confirmed this relationship.[4] Since then several articles have appeared linking not only aortic valvular stenosis but other cardiac disorders including aortic insufficiency, hypertension, and left ventricular outlet obstruction with gastrointestinal bleeding.[5,6] Two specific cases of IHSS have been reported.[7,8]

The radiographic appearance of angiodysplasia is that of a focal collection of a dilated rete of sinusoidal vessels along the antimesenteric border of the colon. The entire lesion may be no more than 3 to 4 mm. in diameter. Characteristically, there is rapid filling of the serpiginous vessels during the arterial phase with a dense stain noted. Early venous filling is commonly seen into an enlarged draining vein. At surgery, however, the lesions usually are not visible unless the overlying bowel mucosa has ulcerated or become atrophic with resultant bleeding. It is therefore probable that angiodysplasia is more common than indicated, the vascular ectasia being concealed by intact mucosa. A recent article by Rogers suggests that colonoscopic diagnosis may be feasible if submucosal vessels are visible.[9]

Following surgical resection, the gross examination is frequently normal and special preparation of the specimen is necessary. The arteries must be injected with silicone rubber or barium gel and then appropriately cleared to demonstrate the abnormal cluster of vessels.[10] Grossly, the appearance has been likened to a coral. Histologically, large submucosal vascular spaces are identified with ulceration and thinning of the mucosa. Our case is unusual in that the pathologic findings were apparent without benefit of latex rubber injection.

The etiology of angiodysplasia has not been determined, although increasing opinion favors an acquired rather than a congenital disorder, particularly as the entity is seen almost exclusively in the elderly. Boss states that isolated aortic valvular stenosis is most frequently seen secondary to a congenital malformation.[7] As congenital malformations may be frequently

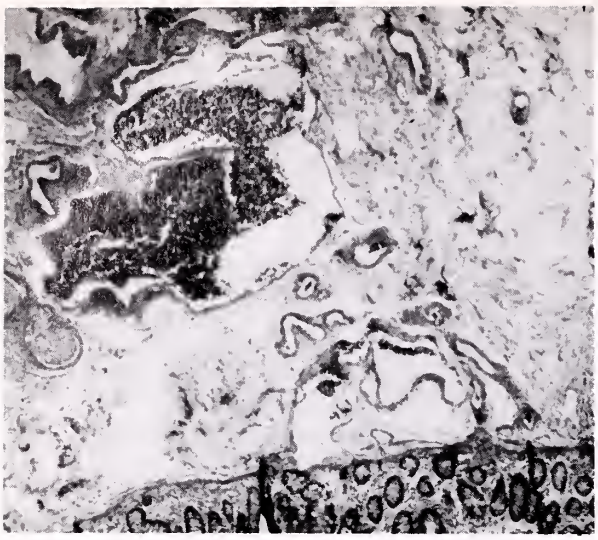

Fig. 2. Histologic section of the right colon. Multiple clusters of large thin walled irregular submucosal vessels are seen. Several demonstrate organized thrombus suggesting chronicity. Barium granuloma is seen in the lower right-hand corner just above the mucosa.

TABLE I

Cardiac Catheterization Data

Position	Pressures (mm Hg)	Mean
RA	A-5 V-3	2
RV	20/3	
PA	20/7	12
PAW	A-6 V-4	5

Simultaneous Pressures (mm Hg)

	Post PVC	Valsalva	Propanolol infusion
LV	260/20	225/50	136/18
AO	120/50	120/80	130/60

Cardiac output - 3.8 L/min.

RA - Right atrium
RV - Right ventricle
PA - Pulmonary artery
PAW - Pulmonary artery wedge
LV - Left ventricle
AO - Aorta

multiple, predisposition to vascular anomalies of the right colon may occur. Baum, however, maintains that submucosal vascular ectasias are more likely secondary to chronic submucosal arteriovascular shunting due to increased intraluminal bowel pressure. LaPlace's law, relating intraluminal pressure and tension as a function of increased diameter, may explain the more frequent involvement of the right side of the colon.[11,12]

Boley suggests that angiodysplasia represents a degenerative process as it is present in a large segment of the population over 60 years of age.[13] It is frequently multiple, and may be the most common etiology of lower gastrointestinal bleeding in the elderly. He feels that bowel dilatation results in chronic transient obstruction to the submucosal veins, resulting in dilatation of the veins, venules, capillaries, and arteries, and thus ultimately leads to incompentency of the precapillary sphincters. This results in an arteriovenous communication. The studies of Boss, Galloway, and McNamara have identified diminished perfusion pressure secondary to valvular obstruction as a common etiologic factor.[7,8,14] Galloway felt that diminished cardiac output results in peripheral vasoconstriction and transient splanchnic hypoperfusion.

Angiodysplasia may therefore represent the end stage or final common pathway of multiple etiologies. Increased intraluminal pressure resulting from constipation, incarcerated hernia, obstructing carcinoma, or other causes may result in chronic transient ischemic bowel disease. Similarly, cardiovascular disease such as aortic valvular stenosis, IHSS, congestive heart failure, myocardiopathy, hypertension, and cardiac arrhythmias may result in diminished cardiac output. Splanchnic vasoconstriction occurs and may result in chronic transient ischemic bowel disease. Causes for vascular ectasia may be additive. One might imagine the not uncommon occurrence of aortic stenosis and constipation co-existing in the elderly patient resulting in angiodysplasia, both processes together critically compromising splanchnic perfusion.

Summary

A case report of IHSS with associated angiodysplasia is presented. The role of cardiac disease associated with angiodysplasia is explored. Unexplained chronic lower gastrointestinal bleeding in a patient with cardiac disease, particularly aortic stenosis, should

prompt vigorous investigation. Visceral angiography remains the definitive test for the diagnosis of angiodysplasia. Vascular ectasia probably represents the end result or final common pathway of multiple etiologies, among them low perfusion states secondary to diminished cardiac output.●

ACKNOWLEDGEMENT

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Acute leukemia presenting as a ruptured spleen*

Report of a Case

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Acute leukemia most commonly presents with symptoms of infection and/or bleeding. Splenic rupture is a well-known, but unusual, complication. Patients presenting with splenic rupture are unusual.[1,2]

CASE REPORT

A 52-year-old male carpenter was in good health until two and one-half weeks prior to admission when he developed low grade fever and malaise. Ten days prior to admission to the Albuquerque Veterans Administration Hospital he "passed out" briefly following a normal bowel movement. Upon awakening, severe abdominal pain was noted and he was hospitalized elsewhere. Examination revealed a pale, disoriented man without obtainable blood pressure. His abdomen was firm and distended. CBC showed HCT 19%, HGB 6.0 gm/dl, WBC 45,000/mm^3 with an increase in mononuclear cells.

Emergency exploratory laparotomy demonstrated a ruptured spleen, which was removed. Postoperative recovery was uneventful. The spleen weighed 530 grams and splenic histology demonstrated infiltration with myelomonoblasts. Sternal marrow aspiration was hypercellular with essentially 100 per cent myelomonoblasts, many with Auer rods.

On the tenth postoperative day he was transferred to the Albuquerque Veterans Administration Hospital for further care. On admission the patient was asymptomatic. Further questioning showed a history of minimal abdominal trauma approximately three weeks prior to his splenic rupture. Examination showed generalized lymphadenopathy (0.5 cm diameter) and a healing abdominal incision. Laboratory data included HCT 39%, HGB 13.1 gm/dl, WBC 18,100/mm^3 (17% segemented neutrophils, 2% metamyelocytes, 3% myelocytes, 10% promyelocytes, 35% blasts (some with Auer rods) 23 lymphocytes, 4 monocytes) platelets 247,000/mm^3. Review of blood and marrow confirmed the diagnosis of acute myelomonoblastic leukemia. The patient was treated with a combination of adriamycin, cytosine arabinoside, vincristine, and prednisone, achieving a remission which currently exceeds ten months.

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Discussion

Rupture of the spleen, presumably spontaneous, has been reported in a wide variety of conditions. They include infectious mononucleosis,[1,3,21] systemic lupus erythematosis,[4] anticoagulant therapy,[5] erythroblastosis fetalis,[6] infective endocarditis,[7] hemangiosarcoma of spleen,[8,22] various malignancies,[9] malaria,[10,11] aortic ball degeneration,[12] polycythemia vera,[13] actinomycosis,[14] plasma cell leukemia,[15] chronic lymphocytic leukemia,[16,17] ceroid storage disease,[18] chronic granulocytic leukemia,[16] and acute leukemia.[19,20]

Ravich and others reviewed 772 cases of acute leukemia and noted six cases of ruptured spleen.[19] Since 1970 we have seen 144 cases of acute leukemia in our institutions with only one case of splenic rupture. There are only a few cases where no history of trauma has been elicited. Ravich and others reviewed fourteen cases of rupture, but only eight of these occurred without apparent trauma.[19]

"Spontaneous" splenic rupture has been reported as a presenting complaint very infrequently and in only a few disorders. These include ceroid storage disease,[18] chronic lymphocytic leukemia,[17] acute lymphocytic leukemia,[2] acute myeloblastic leukemia,[1] hemangiosarcoma,[22] and Hodgkin's disease.[23] According to Dahiya and others delayed posttraumatic rupture of the spleen occurs in fourteen to thirty-five per cent of patients. Fifty per cent of these occur in the first week, twenty-five per cent in the second week, and only ten per cent after one month.[24] Since minor trauma may be forgotten, many cases of putative spontaneous rupture of the spleen, as in our case, may really be delayed rupture after minimal trauma to a disease-enlarged organ.

Summary

Acute leukemia presenting as a ruptured spleen is rare. A 52-year-old man with a two-week history of malaise and fever presented in shock with a history of minimal abdominal trauma three weeks previously. A ruptured spleen was removed and, subsequently, he attained a complete remission and remains well more than ten months later.

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Relapsing fever in New Mexico *

Report of Two Cases

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Robert E. Cutler, MD, Espanola, New Mexico and Albuquerque, New Mexico
and William P. Reed, MD

A man and his wife developed a febrile illness several days after multiple bites from ticks of the genus* Ornithodoros. *The illness was manifested by a relapsing fever recurring several times over a period of four weeks. Diagnostic studies including blood smears for* Borrelia *were unrevealing. Organisms idenitified as* Borrelia *were recovered from the ticks. The epidemiology, diagnostic evaluation, and the treatment of this rarely encountered disease are briefly reviewed.

Relapsing fever was first recognized in the United States in 1844, but is now diagnosed infrequently. The disease, however, may be more common than reports indicate since the diagnosis is rarely considered, the symptoms may resolve without treatment, and confirmatory tests may be falsely negative. The disease has been reported from most parts of the world including many of the western states of this country.[1] During the last decade there were outbreaks of eleven cases in Washington state[2], and twelve proven plus other suspected cases in Arizona near the Grand Canyon.[1] We are not aware of any published reports of the disease from New Mexico.

Relapsing fever is caused by spirochetes of the genus *Borrelia*. The organisms may be acquired from lice, especially in epidemic situations, but endemic tick-born disease is more common in the United States. The usual vectors in the mountains of the Western United States are ticks of the genus *Ornithodoros,* including especially *O. hermsii* which has not previously been recognized in New Mexico. The ticks normally feed on rodents, and are carried into unused cabins which serve as a common point of contact with man. The ticks remain alive for months or years in such locations, and may even pass the spirochetes on to their offspring.[3]

Tourists or hunters sleeping in tick infested areas in the summertime may become infected, and are thus incidentally involved in the epidemiologic cycle of this organism. Two cases which occurred recently in Northern New Mexico are described to illustrate the setting and clinical course of the disease.

CASE REPORTS

On the weekend of July 4, 1976, a 44-year-old man and his 40-year-old wife slept overnight in an abandoned mountain cabin near Cordova, New Mexico. The wife was awakened by the sensation of insects crawling over her body and biting her. She captured a few which were full of blood. Six days later, she abruptly developed fever, shaking chills, headache, backache, nausea and vomiting. The course of her fever is illustrated in Fig. 1. Laboratory evaluation, including cultures of blood and urine, was normal aside from slight leukopenia (white blood count (= 3,900/mm^3 with 73% polymorphonuclear leukocytes, 25% lymphocytes and 2% monocytes). She remained febrile with all the above symptoms for four days with a maximum temperature of 104°. At that time, she was given a single oral dose of 500 mg of tetracycline, which was then discontinued because no bacterial cause for the illness was found. Five ticks were brought to the hospital the day after admission and sent to the Vector-Borne Diseases Division of the Center for Disease Control in Fort Collins, Colorado.

Meanwhile, the husband developed fever and was admitted to the hospital with a similar clinical illness. Cultures of blood and urine were negative, and other laboratory evaluation was unremarkable except the white blood count was 12,100 with 69 PMN, 1 myelocyte, 29 lymphocytes and 1 monocyte. The course of his fever is also illustrated in Fig. 1. He recovered spontaneously with no antibiotic treatment,

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but three days later had a relapse lasting one day. Both patients were discharged home with no symptoms other than profound asthenia.

After being afebrile for eight days, the wife had a relapse with all symptoms as before, although this time they were less severe. Physical examination was normal except for tachycardia and a fever of 103°. The fever and symptoms lasted only two days; a blood smear taken just after she had become afebrile was negative for *Borrelia*.

Ten days after his relapse, the husband came to the emergency room with a second relapse of fever. Based on the epidemiologic data so far available, a presumptive diagnosis of relapsing fever was made even though several peripheral blood smears were negative for *Borreliae*. He was treated with parenteral penicillin, and had no further episodes of fever.

Six days after the first relapse ended, the wife was admitted with her second relapse. Peripheral smears were all negative. Again, based on the epidemiologic data, a presumptive diagnosis of relapsing fever was made, and she was treated with oral tetracycline. The fever promptly subsided. Both patients continued to have significant asthenia for another two weeks. No further febrile episodes occurred, and eventually they regained a state of well being.

Acute phase and convalescent serum samples in both patients were submitted to the Center for Disease Control, and there were no significant titer changes for relapsing fever, leptospirosis, Colorado tick fever, or for the rickettsia that cause Rocky Mountain spotted fever, typhus, or Q fever. The ticks which had been collected in the cabin were subsequently identified as *Ornithodoros hermsii*. They were fed on adult mice, and borrelia-like organisms were later found in blood smears of the inoculated mice.** This information was available only after the patients had recovered. The diagnosis therefore rests on the setting, the typical clinical course, and the isolation of the spirochete from ticks known to have bitten one of the patients.

Discussion

The usual manifestations of relapsing fever are nonspecific and variable as shown in Table 1 which summarizes the data available from several series.[1, 4, 5] After an incubation period of about one week, during which time the patient remains asymptomatic, there is an abrupt onset of fever, headache with retro-orbital pain, and severe myalgia or extremity pain. Confusion as well as nausea and vomiting are common, and a wide range of other manifestations are less common. Sudden defervescence occurs two to five days later, and is followed by six to eight afebrile days during which the patient remains asymptomatic aside from the commonly reported asthenia. One to fourteen relapses may ensue in untreated cases. The severity diminishes with each successive relapse, and the febrile periods become shorter and the apyretic phases longer. The diagnosis is usually made on epidemiologic grounds, and often only in retrospect from the characteristic fever curve. The phenomenon of periodic relapses also occurs in malaria, and in both diseases the host responds by manufacturing antibodies that aid in the destruction of most organisms. A few escape destruction to survive through afebrile periods during which they change their surface antigens. The host has no immunity to the new set of antigens, so the organisms can proliferate and cause symptoms all over again.[3] Eventually, the host can develop immunity against all possible antigenic forms, and spontaneous cure occurs.

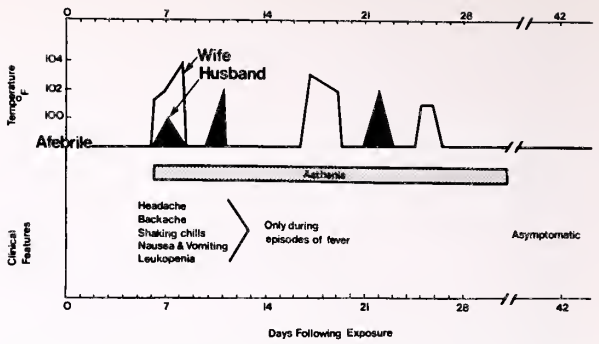

Fig. 1. Clinical features of the two cases. The tick bites which are thought to have initiated the infections occurred on day 0.

Ordinary bacteriologic technics are not helpful since the organisms do not grow on routine culture media. The most common way to make a definitive diagnosis is by seeing the large spiral borrelia in thick blood smears stained with Giemsa or other routine stains (Fig. 2). The prevalence of positive smears varies in different reports, but an overall estimate is that about seventy per cent can be expected to be positive. Additional cases may be identified by inoculation of the patient's blood into experimental animals and observing them for infection.

TABLE I

Symptoms and Signs Found in Relapsing Fever

High Fever with sudden onset	Expected
Severe headache or eye pain	Expected
Severe myalgia or pain in extremities, lumber region and neck	Expected
Alteration of sensorium	Frequent
Nausea, vomiting, or abdominal pain	Frequent
Cough, dyspnea, cyanosis	Infrequent
Tender hepatomegaly and splenomegaly	20-70%
Cardiomegaly, murmurs, heart failure	0-30%
Tender adenopathy	Infrequent

** Studies reported by Dr. Richard Eads of the Vector Born Diseases Division of the Center for Disease Control in Fort Collins, Colorado.

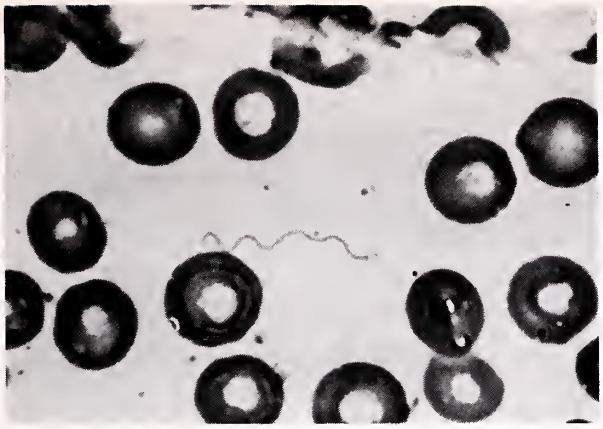

Fig. 2. **Borrelia** *in a peripheral blood smear stained with giemsa. Courtesy Jack S. Remington, MD, Palo Alto Medical Research Foundation, Palo Alto, California.*

Other laboratory tests are variable and less helpful (Table 2). The erythrocyte sedimentation rate is often elevated. Specific serologic tests may become positive but are difficult to perform and are of little value early in the course of the disease. A false positive serologic test for syphilis may occur, and agglutinins may appear for proteus OXK, but not for OX2 and rarely for OX19.

Relapsing fever can be confused with other infections. In the cases described, the more common pyogenic infections were ruled out largely by the absence of symptoms referrable to specific organs or types of infection, and by negative bacteriologic studies. The illnesses were initially compatible with Colorado tick fever, especially in the husband who had only a brief afebrile period between the first two episodes as may occur in Colorado tick fever. This was ruled out by the longer afebrile period in the wife, and by the occurrence of a third episode. The tick that was identified is not a vector for Colorado tick fever. The absence of a characteristic rash ruled out Rocky Mountain spotted fever.

Five diseases may be transmitted by ticks in the Rocky Mountain region: Colorado Tick Fever, Rocky Mountain spotted fever, tick paralysis, tularemia, and relapsing fever. The ticks responsible for the first four of these illnesses are hard ticks of the genus *Dermacentor*. After biting they characteristically attach firmly for prolonged periods. The soft ticks, *Ornithodoros,* that transmit relapsing fever bite only briefly, often painlessly, usually at night, and may be erroneously referred to as bedbugs—thus, the patient may not recognize that he has been bitten by a tick.

TABLE 2

Laboratory Findings in Relapsing Fever

Blood smear for spirochetes	70%
Animal inoculation	86% positive
False serologic test for syphilis	4.5% positive
Erythrocyte sedimentation rate	Frequently elevated
White blood cell count and differential	Nonspecific and variable

Although eventual recovery is the rule, relapsing fever may be fatal, or it may be complicated by iritis, iridocyclitis, or neuropsychiatric, cardiorespiratory and hemorrhagic problems.[4, 5] Some of these complications appear to be more likely after the third relapse.

Because of the variable clinical course of relapsing fever, evaluation of treatment is difficult. Arsenicals, sulfonamides, and antibiotics have been used in the past, and it appears that penicillin, streptomycin, chloramphenicol and tetracycline can be effective in terminating relapses and preventing subsequent ones. Tetracycline is considered now the drug of choice in doses of 500 mg. p.o. every 6 hours for 5-10 days in adults, but the other drugs may be used in children and pregnant women.

Prevention of relapsing fever is mainly through avoiding exposure to arthropod vector attacks and using protective clothing and perhaps insect repellents. Elimination of the *Ornithodoros* tick is practically impossible, but insecticides may be used in sites such as cabins where infections have been acquired.●

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Aseptic necrosis in systemic lupus erythematosus

Early Detection By Radionuclide Scan

Report of a Case

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Aseptic necrosis of bone is a well known complication of systemic lupus erythematosus (SLE) or corticosteroid therapy.[1] We wish to report a case of aseptic necrosis detected by radionuclide scanning before the appearance of diagnostic changes on conventional roentgenograms or clinical signs and symptoms.

CASE REPORT

A 21-year-old male fulfilled the preliminary Arthritis and Rheumatism Association criteria for SLE.[2] He has been treated with azathioprine 100-150 mg per day and prednisone 30-80 mg per day for the last three years depending on clinical activity.

On December 20, 1974 he complained of slight pain in the right hip which he attributed to minor trauma; there were no objective signs of joint disease on physical examination. He returned January 1, 1975 complaining of pain over his right buttock. Physical examination showed decreased flexion of the right hip with loss of 15% extension and a slight decrease of external rotation. There was no pain or swelling in other joints. On February 28 a 99m Tc-pyrophosphate scan and x-rays of the hips and ankles were obtained. There was increased uptake over the right femoral head and right ankle joint. Conventional roentgenograms showed aseptic necrosis of the right femoral head, and normal ankles (Fig. 1 and 2). Gallium-67 citrate and 99m Tc-pertechnetate radionuclide scans performed on March 3 showed increased uptake over the right ankle and femoral head (Fig. 3 and 4). On April 11 the patient complained of pain in his right ankle and tenderness was localized over the talus, but there was no synovial swelling or effusion. Roentgenograms of the ankles were normal. On August 26 the patient reported increased pain and limited motion in his right ankle, and roentgenograms at this time were interpreted compatible with aseptic necrosis (Fig. 5).

Radiographic followup on May 3, 1976 showed development of aseptic necrosis in his left ankle and right hip. The patient has shown no signs of septic joints, lymphomas, or active SLE synovitis.

Discussion

Initially this case was a diagnostic dilemma. it was thought that the patient's pain was not SLE-related arthritis[3] or septic arthritis because no synovial swelling or pain was present over the right ankle. In addition, when the right ankle became symptomatic, the pain was greatest over the talus and not over the

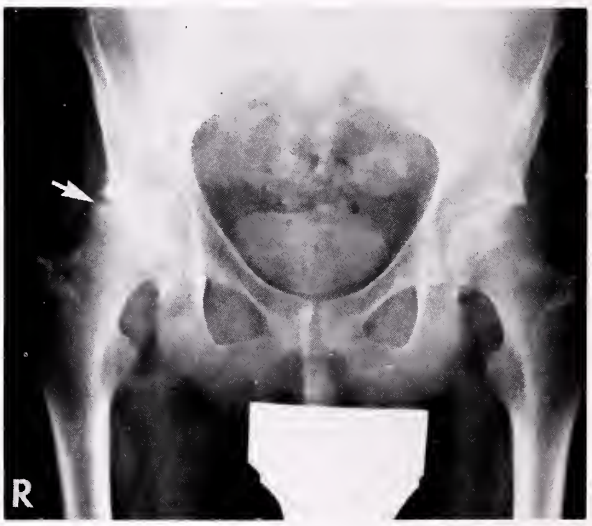

Fig. 1: AP pelvis film, February 1975 shows flattening and irregularity of right femoral head compatible with aseptic necrosis.

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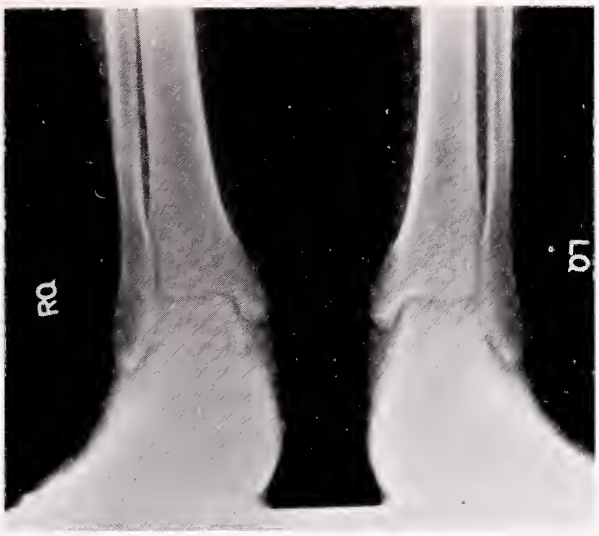

Fig. 2. AP, both ankles, February 1975: negative.

Fig. 3. Gallium-67 citrate scan of pelvis, March 3, 1975, shows increased uptake in the right femoral head.

Fig. 4. Gallium-67 citrate scan of ankles March 3, 1975, shows increased uptake over the right talus and ankle.

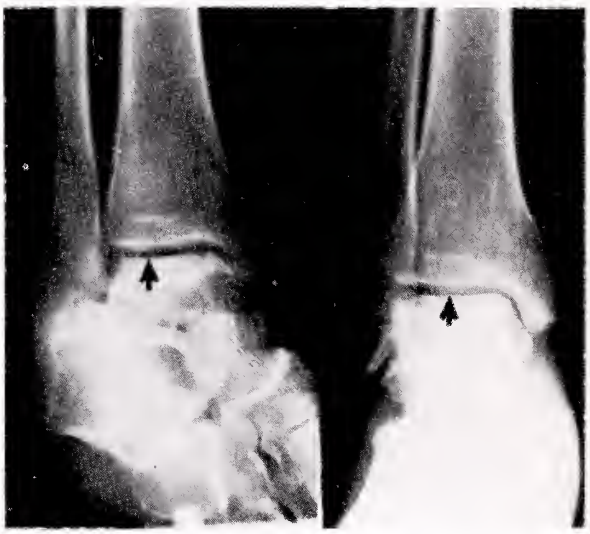

Fig. 5. AP, oblique, right ankle films, August 26, 1975, demonstrate aseptic necrosis with flattening and mottling of talus. White arrow indicates radiolucent subcortical line.

synovium. There were no signs of generalized activity of SLE either on physical examination or in laboratory studies. This patient is of interest because positive findings on radionuclide scans of the right ankle antedated changes on conventional x-rays by as much as six months and clinical signs by one and one-half months.

It is well recognized that technetium 99m radionuclide bone scans are positive in a variety of rheumatologic disorders such as rheumatoid arthritis, gouty tophi, osteoarthritis, polymyositis, and ankylosing spondylitis.[4–9] Early aseptic necrosis often shows decreased uptake paralleling the decreased vascular flow while the later, remodeling stage, shows increased uptake of bone scanning agents.[10] Bone scans have been reported as diagnostic in aseptic necrosis in fracture patients, renal transplant patients and Legg-Perthes disease.[10,11] Aseptic necrosis in SLE patients on steroids can now be added to this list. This is the first case to

our knowledge in which Gallium-67 citrate accumulation has pre-dated roentgenographic changes of aseptic necrosis. The long interval between the three positive radionuclide scans and the diagnostic x-ray findings suggest that these techniques are useful in early detection of this disorder.

Summary

A patient with systemic lupus erythematosus treated with steroids showed increased uptake of three different radionuclides in an ankle before the appearance of clinical findings or radiographic evidence of aseptic necrosis. Since there was increased uptake of Gallium-67 citrate, the differential diagnosis of an abnormal accumulation of Gallium in the area of a painful joint should include aseptic necrosis.●

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Microtia in New Mexico*

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and Jon Aase, MD, Albuquerque, New Mexico

New Mexico has the largest series of patients with microtia in the United States. The data obtained in review of this series assists care of microtia patients.

One hundred sixteen (116) patients with microtia have been seen in New Mexico. This is the largest series in the United States and therefore has been reviewed in depth.

Microtia is defined as small (micro) ear (otia). It is manifest as a spectrum from minimal distortion and diminution of ear architecture (Grade I) to almost total absence of any ear structures (Grade III).

Characteristics of the Population

The characteristics of the population are outlined in Table I. The incidence figures for the white population are in keeping with those of other studies, while the incidence in the American Indian is some eight times higher. This suggests that genes associated with the production of microtia are carried in high frequency in the Indian group.

The higher proportion of affected males in this series may be caused in part by reluctance of affected girls to present themselves for examination, and by the greater ease with which the defects may be concealed by feminine hair styles. Still, the magnitude of the difference between the sexes suggests a real predilection for males, which would be in keeping with other reports of this condition.

The racial and sex distribution of microtia in our series would fit well with this "multifactorial hypothesis" of causation. This implies that multiple genes of small individual effect combine with presently unknown environmental factors to allow the maldevelopment of a specific organ or system. Those congenital disorders thought to be of multifactorial origin have several factors in common: usually only a single developing body structure is affected; there is a consistent predilection for one sex; the incidence varies in different racial groups; and the recurrence risk for unaffected parents of an affected child is 3-5 per cent. All of these criteria seem to be met in the present series where the recurrence risk is 5.7 per cent.

Characteristics of the Deformity

The characteristics of the deformity itself are depicted in Table 2. In this series, as in other reported surveys, microtia occurs more commonly on the right side. Table 2 also indicates a predominance of more severe microtia (Grade III) in this series, possibly reflecting a failure of referral of cases with milder deformity.

Over 90 per cent of these patients had auditory canal atresia and a hearing deficit in their microtic ears. Eleven per cent also had hearing loss in their non-microtic contralateral ear. These patients need to be evaluated to determine whether they need hearing aid(s) and followed closely to detect and treat any otitis or other ear disease which could further reduce their hearing.

Forty-six per cent of these patients with microtia also had other anomalies. Twenty-six per cent were classified as having first and second branchial arch syndrome which was not unexpected since the ear is derived embryologically from the first and second branchial arches. Other associated anomalies affected virtually every other body system (Table 3). This emphasizes the importance of a thorough general examination.

*For Reprints, write Dr. Tegtmeier at 2701 West 84th Avenue, Suite 201, Westminster, Colorado 80030.

TABLE 1

Characteristics of Population

Race	Number	Incidence of Microtia
Indians	50	1:1900
Spanish-Americans	39	1:9000
Anglo	27	1:16000
Sex	**Number**	**Percentage**
Male	69	60%
Female	47	40%

TABLE 2

Characteristics of the Deformity

Laterality	Number	Percentage
Right	74	66%
Left	33	29%
Bilateral	5	5%
Grade	**Number**	**Percentage**
I	16	18%
II	27	30%
III	47	52%

Since genitourinary maldevelopment has been reported in association with ear anomalies, intravenous pyelography was performed in 26 patients in this series. Only 2 (7.7 per cent) showed evidence of minor genitourinary anomalies, and neither had been symptomatic. Routine IVP's, therefore, do not seem indicated, though urinary symptoms should be investigated more aggressively than in non-microtic patients.

Ear Reconstruction

The major concern of these patients is the stigma of the birth defect itself. We reviewed the ear reconstruction efforts among these microtia patients to see if the results could be improved.

Simple rearrangement of tissues of local flaps in conjunction with small cartilage implants can be adequate to reconstruct minor degrees of microtia. In more severe deformities, however, these methods produced poor results even after many (average 6.4) operations.

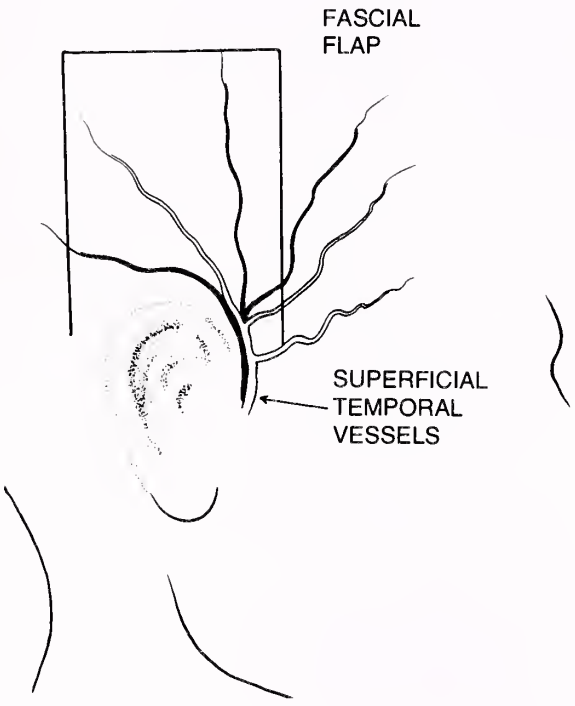

Fig. 1. Fascial Flap with superficial temporal vessels.

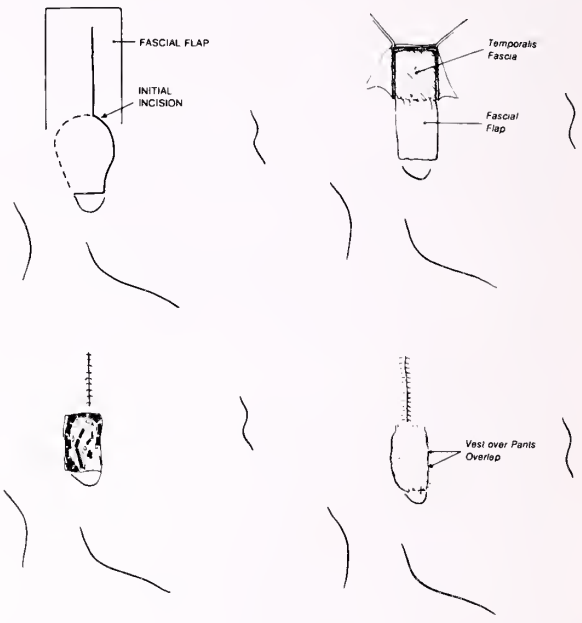

Fig. 2. Operative technics for use of fascial flap in ear reconstruction.
a. Initial incision.
b. Development of fascial flap.
c. Turning indigenous skin back into sulcus and placement of proplast-silicone implant.
d. Turning fascial flap over implant and closing suture line with vest-over-pants closure.

TABLE 3

Associated Congenital Anomalies

Data regarding associated anomalies was available on one hundred (100) patients. Forty-six percent of the patients has associated anomalies. Several had more than one anomaly. Twenty-six percent of the patients had one or more signs of the first and second branchial arch syndrome, i.e., mandible hypoplasia, facial weakness, hypoplastic parotid, or macrostomia.

Head and Neck

Ipsilateral Hypoplastic mandible	22)	First or Second
Ipsilateral Facial Weakness	10)	Branchial Arch
Ipsilateral Hypoplastic Parotid	3)	26 Patients
Macrostomia	5)	(26%)
Preauricular Skin Tags	8	
Cleft Lip and/or Palate	4	
Prominent Opposite Ear	1	
Cupped Opposite Ear	1	
Partial Alopecia	1	
Cranial Asymmetry	1	
Treacher Collins Syndrome	1	

Upper Extremity

Syndactylism	1
Hypoplastic Digits	1
Abnormal Hand Creases	1
Fusion Ulna and Radius	1
Hypoplactic Clavicle	1

G. U. Anomalies

Duplicated Ureter	1
Calyceal Diverticulum	1
Undescended Testes	2

Lower Extremity

Polydactylism Foot	1
Club Foot	1

Other

Tetralogy of Fallot	1
Spina Bifida	1
Meningocele	1
Dwarfism 2^{o} Cartilage Hypoplasia	1
Imperforate Anus	1
Mesenteric Cyst	1
Rib Anomalies	1

Thirty-one patients had Cronin ear reconstruction[3] with silicone implants and postauricular skin cover. The aesthetic results of these reconstructions were generally good. However, 55 per cent of these implants became exposed and 53 per cent of the implants which became exposed also became infected and had to be removed.

We tried to determine why some exposed implants were lost and others were salvaged. If patients were available for and cooperative with close followup, the exposures could be detected and treated easily. Early antibiotics, excision of the exposed implants, and reestablishment of skin cover salvaged many implants. However, patients who had a more rugged lifestyle and were not available for close followup had more exposures and lost almost all exposed implants.

We concluded that the durability of the repair depends on the lifestyle of the patient and the quality of followup.

We have sought to improve the methods available for ear reconstructions, by developing a new cover, a new implant, and a new ear dressing.

The need for a thin, tough, abundant, readily available, hairless cover lead to consideration of the galea aponeurotica and the lateral superficial fascia which is in continuity with the galea. This fascia is tough, being primarily composed

of collagen. By including the superficial temporal vessels with the fascia, an arterial pattern flap can be created which is large enough to cover the entire ear (Fig. 1). This flap has to be covered with some form of epithelium, i.e., a skin graft. The fascia and skin graft together are usually thinner than the postauricular skin commonly used in ear reconstruction. Experimentation with dog and Rhesus monkey models proved the fascial flap could be used in ear reconstruction.[1]

Proplast is a porous material of carbon and teflon with a low extrusion rate, probably due to the ingrowth of tissue into the pores. We designed an implant using a silicone core for resilience and a thin proplast coverage in all prominent portions. This proplast-silicone implant has been used to try to reduce the extrusion rate in our population.

Room-temperature-vulcanizing silicone foam is used as the ear dressing.[2] It is vulcanized directly on the reconstructed ear to form an exact negative image. It is firm enough to reduce edema but yet has enough resiliency that it does not compromise circulation.

These methods (Fig. 2) have now been used in 16 patients, and produce quite satisfactory ears (Fig. 3) with fewer operations than most previous methods. The exposure . and loss rates have been reduced to 27 per cent and we feel will be reduced even further with the newly implemented vest-over-pants closure of galeal flap to skin. The proplast implant, however, does lend a dark hue to the skin if used in light skinned individuals, and we look forward to the development of a white proplast-like material.

Fig. 3. Ear reconstructed with fascial flap, proplast-silicone technic.

Conclusions

Maldevelopment of the external ear (microtia) occurs with more frequency in American Indians, in males and on the right side. Associated abnormalities are relatively common but renal anomalies may not be as frequent as previously supposed. For isolated microtia, the recurrence risk is in the neighborhood of 5 per cent.

New surgical procedures are available which can provide good cosmetic results with a minimal number of procedures and a low failure rate.

Summary

We have reviewed a series of 116 microtia patients seen in New Mexico, their clinical management, and efforts at ear reconstruction among these patients. New surgical technics which promise improved cosmetic and functional results in reconstruction of the ear, are described.●

CREDIT

To Plastic and Reconstructive Surgery Journal for use of figures.

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[1] Tegtmeier, Ronald E., and Gooding, Richard A., "The Use of a Fascial Flap in Ear Reconstruction.", *Plast Reconstr Surg*, September, 1977.

[2] Tegtmeier, Ronald E., "A Silicone Foam Ear Dressing.", *Plast Reconstr Surg* July, 1977.

[3] Cronin, T.D.: Use of a Silastic Frame for Construction of the Auricle. *Symposium on Reconstruction of the Auricle*. Educational Foundation of the American Society of Plastic & Reconstructive Surgery, C.V. Mosby, St. Louis, Mo.:33, 1974.

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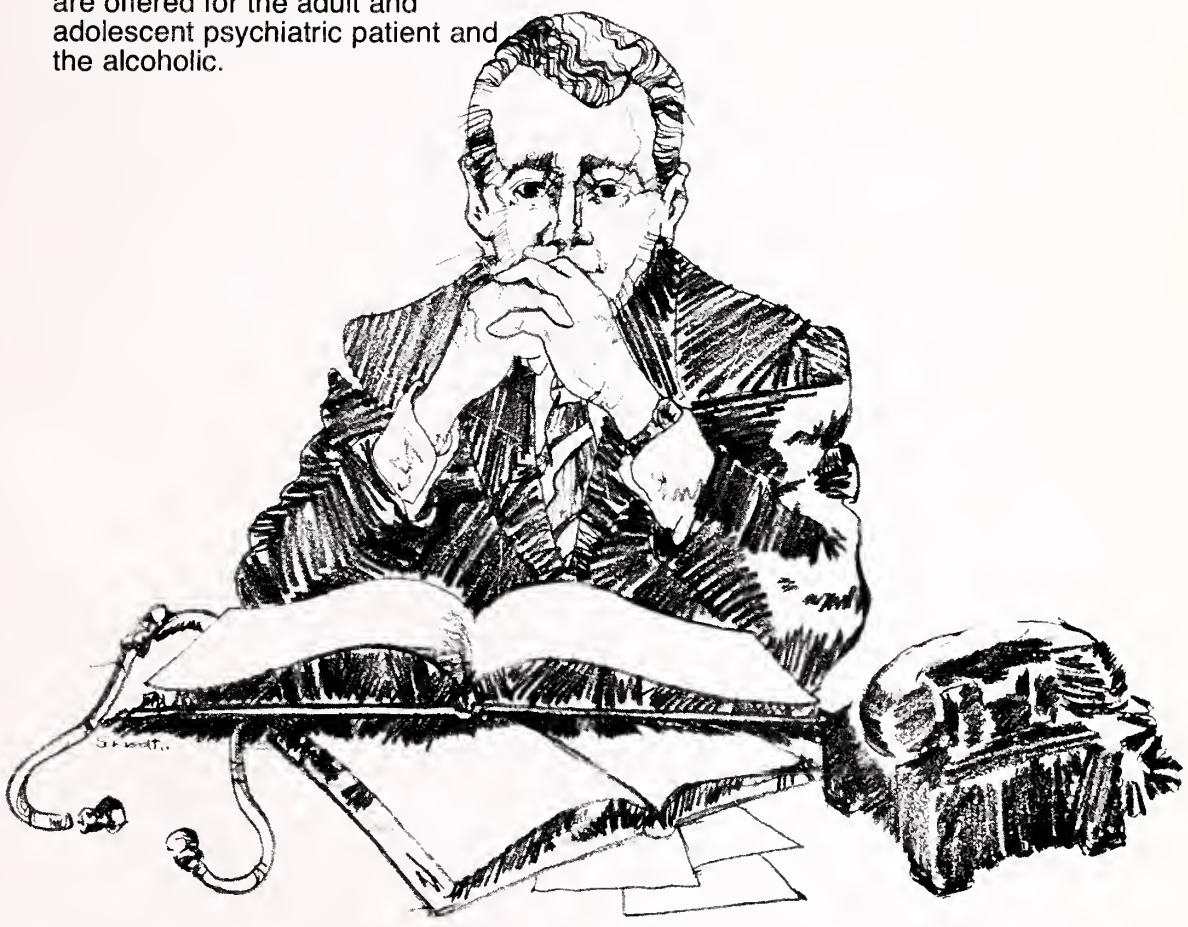

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Wednesday, January 25, 1978

Wednesday, January 25
Afternoon Session: 4:30 pm-6:30 pm
Topic: Carcinoma of the Bronchus-The Problem
Moderator: Weston Reynolds, MD
Etiology-Geno Saccommano, MD
Epidemiology-John Berg, MD
Environment-Paul Kotin, MD

Thursday, January 26
Morning Session: 7:30 am-9:30 am
Topic: Diagnosis
Moderator: F. B. "Bing" Johnson, MD
Jeffrey D. Rudikoff, MD
Lawrence Repsher, MD
Geno Saccomanno, MD

Afternoon Session: 4:30 pm-6:30 pm
Topic: Treatment by Surgery and Radiation
Moderator: W. Gerald Rainer, MD
Thomas Ferguson, MD
Robert Parker, MD

Friday, January 27
Morning Session: 7:30 am-9:30 am
Topic: Chemotherapy
Moderator: David Garfield, MD
Robert Livingston, MD
Topic: Immunotherapy
Thomas Ferguson, MD
Jan L. Gray, MD

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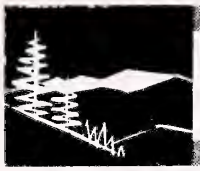

COLORADO

Boswick Named Secretary

John A. Boswick, Jr., MD, Professor and Chairman of the Hand Surgical Service at the University of Colorado Medical Center has been re-elected Secretary of The American Association for the Surgery of Trauma. The Association is a 39-year old medical organization dedicated to the problems of care of trauma patients.

Asthma Center Receives Contract

Free care for 150-200 asthmatic children, including transportation, will be made available by the National Asthma Center under a $226,000 contract awarded by the National Institute of Heart, Lung, and Blood. Only three awards were made under the direction of the National Institutes of Health.

Thomas Creer, MD, behavior science head at the Center, will administer the contract which will focus on technics developed in years of caring for resident patients in assisting less severely ill patients. The contract's aims are to teach self-control skills to asthmatic children as a self-management process, avoiding stimuli to attacks and complying with medication instructions, so that they will respond properly with some measure of control over its severity. The effort to correct certain misconceptions about asthma is also given stress.

Farrington Speaks in Meeting

John Farrington, MD, Boulder, was among those speaking at a two-day workshop in Denver, October 5 and 6, entitled "An Examination of Health Care Policy in the United States," and sponsored by the Medical Care and Research Foundation.

Denver Doctors Direct Workshops

Among the leading medical laboratory experts who conducted more than 180 educational workshops and seminars during the joint meeting of the American Society of Clinical Pathologists and the College of American Pathologists from October 20 through 28 in Las Vegas, Nevada were four staff members of Denver medical facilities. These were Benny F. Mertens, MD, St. Joseph Hospital; Thomas A. Merrick, MD, Presbyterian Medical Center; J. Kenneth McLatchey, Ph D., National Jewish Hospital, and Mr. John H. Waters of St. Anthony Hospital Systems.

This team directed five different workshops for the members of the meeting.

Another Denver medical laboratory expert will direct two workshops at the joint meetings. Elmer W. Koneman, MD, will lead one involving laboratory diagnosis of fungus diseases, as well as contributing time to other aspects of pathology.

Two doctors from the University of Colorado School of Medicine, Steven G. Silverberg and Sara D. Winter, are others involved in the direction of workshops at the meet-

Basye Named ACFP Director

Charles Basye, MD, Fort Collins, Colorado, has been elected to a three-year term as a director of the American Academy of Family Physicians. Elections were held in October during the annual meeting in Las Vegas, Nevada.

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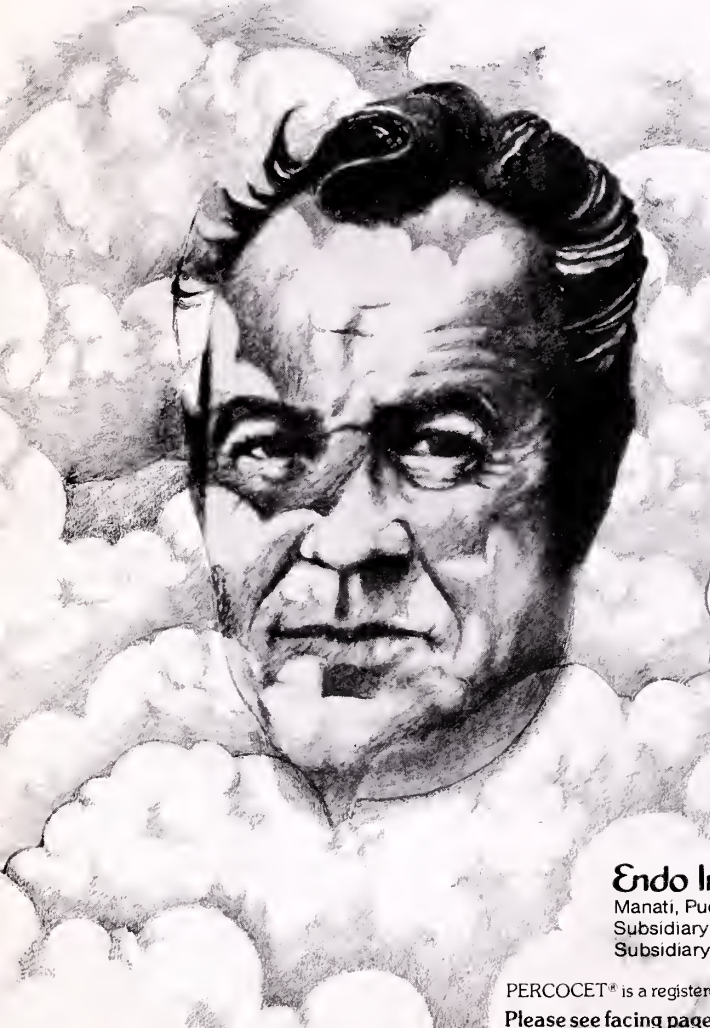

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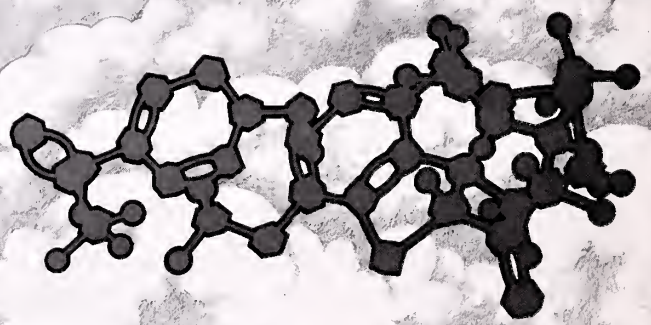

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OBITUARIES

Colorado

Doctor Harmon P. Brandenburg died July 12, 1977 at Wheat Ridge, Colorado. He was 90 years of age. Born in Washington, D.C. July 24, 1886, he came to Denver and in 1910 was graduated from the University of Colorado School of Medicine.

After serving in general practice in Lander, Wyoming, in 1914 he went to Cook County Hospital in Chicago as assistant radiologist. A year later he returned to Denver and opened an office for the practice of radiology. He was radiologist for Mercy Hospital, Beth Israel Hospital, Denver General Hospital, and National Jewish Hospital during his years of active practice. He retired in 1951.

In addition to membership in the American Medical Association he belonged to the Denver and Colorado Medical societies, the American Radium Society, the Colorado Radiological Society, and was a Fellow of the American College of Radiology.

He was married to Louise Tourtellotte in 1912. She died in 1924. In 1935 he was married to Julia A. Grantham who died in 1971. He is survived by two sons, Dr. Frederick H. Brandenburg of Denver and John F. Brandenburg of Delevan, Wisconsin.

Doctor Charles A. Cassidy of Monte Vista, Colorado, died Monday, August 15, 1977 in Denver after a long illness. Born January 8, 1917 in Las Vegas, New Mexico, he was graduated from St. Louis University School of Medicine in 1942. He interned at St. Johns Hospital, St. Louis, Missouri.

After serving a residency in surgery at Mercy Hospital in Denver, he opened a practice in Monte Vista, Colorado.

He was an active member of the San Luis Valley and Colorado Medical societies, and of the American Medical Association.

He is survived by his widow and a son, Charles A. Cassidy, Jr., Whittier, California, and two grandchildren.

Doctor William A. Day died July 16, 1977 in Colorado Springs following a brief illness. Born March 18, 1911 in Oshkosh, Nebraska, and educated in that state, he came to Colorado in 1939, establishing a general practice in Julesburg, Colorado where he remained until 1948.

At that time he took a year of graduate training in Proctology at the Ferguson Clinic and Hospital in Grand Rapids, Michigan, after which he opened an office for the practice of Proctology in Colorado Springs.

He is survived by his widow, Mrs. Jane Day, two sons, Dr. Jefferson Day, Eureka, California, and Stephen Day of Alameda, New Mexico, and a brother, Robert Stanley Day, Tampa, Florida.

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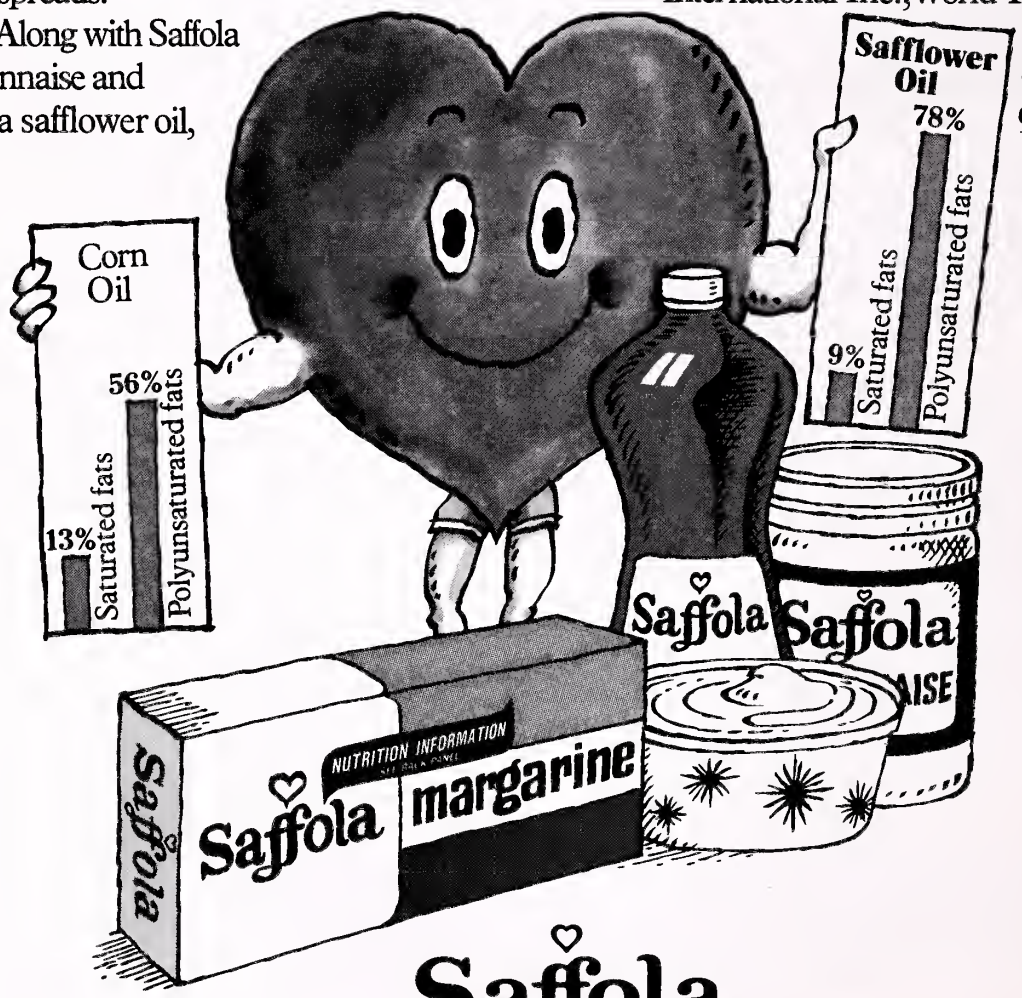

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New Mexico Medical Society

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